

STUDIES IN THE AGRICULTURAL AND FOOD SCIENCES

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AND FOOD SCIENCES

Plant Breeding for Pest and Disease Resistance

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PREFACE

It has been estimated that more than 50 per cent of the world's total crop production is lost each year through the activities of pests and diseases of plants, in spite of the many control measures that are employed. The importance of reducing this loss, particularly in the developing countries, hardly needs emphasizing and additional control measures are urgently needed. The main purpose of this book is to review the part played by resistant varieties in reducing damage by pests and diseases in the past, and to assess the potential value of breeding for resistance.

The book is arranged in six sections, each of which can be read independently; this arrangement has, inevitably, led to some duplication between sections but repetition has been kept to a minimum. The first section is concerned with general principles of pest and disease control by resistant varieties. The following four sections deal with fungal diseases, diseases caused by viruses, bacteria and other micro-organisms, animal pests and parasitic weeds, respectively. Within each section there are chapters relating to the special features which affect breeding for resistance to the different kinds of parasites. These features are illustrated by reference to specific examples from some of the world's most important agricultural and horticultural crop plants. The examples have been chosen to demonstrate successes and failures in breeding for resistance, with suggested explanations for the varying degrees of success that have been achieved. An attempt has been made to pinpoint areas where our knowledge is inadequate, and where a greater effort and more research facilities seem to be justified.

The final section comprises some general conclusions and summarizes the author's view on the future prospects of breeding for resistance.

Each of the main sections contains several hundred references to the original work that is mentioned in the text and the book should, therefore, be an important source of references for many years to come.

It is hoped that the book will be useful both to the plant breeder and to agricultural specialists in many other disciplines, who may not always appreciate the full potential of breeding for resistance. In addition, it should be a useful reference book for postgraduate and undergraduate students working in many areas of applied biological science.

GORDON E. RUSSELL

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GORDON E. RUSSELL

I

THE CONTROL OF PESTS AND DISEASES

Economic Importance of Pests and Diseases

Pests and diseases of agricultural crops are as old as agriculture itself. The effects of mildew diseases and of insect pests, including locusts, are recorded in the Old Testament. Cereal rusts are known to have been important during the Roman period and exhaustion of the soil at this time was probably caused by parasitic nematode pests. The Romans invoked the aid of gods to keep their crops free from disease and epidemics were attributed to the wrath of the gods. It was not until many centuries later, after the general acceptance of Pasteur's germ theory, that diseases of crop plants were recognized as being caused by parasitic organisms. The specific causal agents of diseases such as potato blight or cereal rusts were identified as fungal pathogens only in the latter half of the nineteenth century. Such discoveries helped to end the fatalistic attitude towards the ravages of pests and diseases and stimulated attempts to discover methods of controlling them.

One of the best-known examples of a catastrophic epidemic of a plant disease is the outbreak of potato blight (late blight) which occurred in 1845 and 1846 in western Europe. This disease, caused by a fungus (*Phytophthora infestans*) which has airborne spores, was probably introduced into Europe from Mexico in the early 1840s. Small outbreaks of blight occurred in parts of France, Belgium and England in 1844, but the dry summer of that year did not favour the spread of infection. In July 1845, however, the disease became widespread in northern Europe where it caused severe damage to the potato crop, particularly in Belgium and Ireland where at least 40 per cent of tubers rotted as a result of infection. In 1846 the disease attacked the potato crop in Ireland at an earlier stage of growth, and spread so rapidly that all plants in many crops had been killed by the beginning of August. The potato was the staple food of the mainly peasant population of Ireland and widespread famine followed the blight epidemics; as a direct result the population of Ireland declined from 8.2 million in 1841 to 6.2 million in 1851, either from death resulting from starvation or from emigration to Britain or North America. The impact of potato blight in other parts of northern Europe was also considerable, although generally less severe than in Ireland.

Although it is unusual for a single disease to affect the course of history in this catastrophic manner, many diseases other than potato blight have had

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serious economic consequences (Klinkowski, 1970). Some such disease epidemics have been caused by fungi, including blue mould of tobacco (*Peronospora tabacina*) in Europe in 1960, downy mildew of hops (*Pseudoperonospora humuli*) in Central Europe in the 1920s, Dutch elm disease (*Ceratocystis ulmi*) in Europe and North America in recent years, rusts of cereal crops (*Puccinia* spp.) in many parts of the world, coffee rust (*Hemileia vastatrix*) in Sri Lanka (Ceylon) and brown spot of rice (*Helminthosporium oryzae*) in Bengal. Other disease epidemics have been caused by viruses including swollen shoot disease of cocoa, sugar cane mosaic and curly top of sugar beet, or by bacteria such as the bacterial blights of cotton and rice. The resultant losses in economic terms are impossible to estimate accurately because the severity of a disease varies greatly from place to place and from year to year. However, it has been shown experimentally that these losses can be very considerable. For example, virus yellows can reduce the yield of sugar beet by more than 3 per cent for every week that a plant shows symptoms (Hull, 1961) and losses of more than 25 per cent attributable to virus yellows have often been recorded in farm crops. In the USA in 1935, the loss of yield in wheat due to the stem rust fungus in three States alone was estimated to be between three and four million tons of grain. Later outbreaks of this disease in 1954 and 1957 caused estimated losses of 45 million bushels and 150 million bushels of grain respectively in western Canada. Wheat, barley, maize, potatoes and tomatoes were calculated to have lost, respectively 6.3, 5.5, 8.6, 18.2, and 12.6 per cent of their potential yields in the USA from diseases in 1939 (Ordish, 1952). In 1937, insect pests in the USA were estimated to have caused losses running into millions of dollars in many crops, particularly cotton (boll weevil and earworm), maize (earworm), wheat (chinch bug and Hessian fly), potatoes (Colorado beetle and leaf hoppers) and tobacco (budworm and hornworm). More recent estimates suggest that losses from pests and diseases are even greater than were suggested by Ordish. Cramer (1967), for example, calculated that world-wide losses from diseases and insect pests in wheat were 9.1 and 5.0 per cent of the potential yield respectively. Corresponding figures for diseases and pests were 7.8 and 3.8 per cent for barley, 9.4 and 12.4 per cent for maize, 21.8 and 6.5 per cent for potatoes, and 8.9 and 26.7 per cent for rice. Cramer estimated that average world-wide losses for the main agricultural crops were 11.8 per cent for diseases and 12.2 per cent for insect pests. The average combined losses caused by diseases, pests and weeds were put at 33.7 per cent. This figure is considerably less than recent estimates that more than half of the world's potential crop production is lost by the action of diseases and weed and insect pests.

Although it is impossible to quantify these losses accurately, the above estimates emphasize the enormous damage that is caused by pests and diseases. The development of more effective methods of controlling pests and diseases in the major crop species is probably the most urgent and daunting task facing the agricultural scientist today.

Methods of Controlling Pests and Diseases

It is convenient to group control methods into three main categories:

(1) avoidance of pests and diseases, (2) direct control measures, and (3) biological control.

AVOIDANCE OF PESTS AND DISEASES

Measures to avoid pests and diseases can be considered briefly under the following nine headings.

Quarantine

Legislation can prevent the introduction of infected plant material, including seeds and other material for propagation, into areas where a pest or disease is absent. Most countries have laws controlling the importation of plant material or its movement from one part of the country to another. These regulations, which have been discussed by Wheeler (1969), have been successful in preventing or delaying the spread of many important pathogens and pests. For example, the Colorado beetle (*Leptinotarsa decemlineata*), which is an important pest of potatoes in many countries, has so far been denied a foothold in the UK, mainly because of stringent quarantine and legislative measures. Such quarantine measures are likely to become less effective in the future, however, because fresh plant material, either for food or for experimental purposes, can now be transported much more rapidly and in better condition than in the past. In addition, plant breeders seeking to exploit germplasm from exotic species in their breeding programmes, have sometimes unwittingly introduced exotic pests and diseases with experimental plant material. Quarantine must therefore be generally considered as a first line of defence which will almost certainly be breached sooner or later.

Legislation has been used successfully to control the spread of certain pests and diseases within a particular country or region. For example in England a 'Wart Disease Order', which prohibited the planting of potato varieties that were very susceptible to the causal fungus *Synchytrium endobioticum*, was introduced in 1923. The Order, which was amended in 1941 and 1973, also controls the movement of potato tubers from infected areas and requires the destruction of infected tubers. These measures have been strictly enforced and the disease has become less widespread and damaging as a result.

Apple and pear trees can be attacked by fireblight, a disease caused by a bacterium, *Erwinia amylovora*. A 'Fireblight Disease Order', first introduced in England in 1958 and amended in 1960 and 1966, requires that any known or suspected cases of fireblight must be reported to the authorities. The Order, which also prohibits the propagation of very susceptible varieties, has undoubtedly helped to control the spread of fireblight disease in the UK.

Cropping on land which is known to be heavily infested with the beet cyst nematode *Heterodera schachtii* is controlled in England by the terms of the 'Beet Eelworm Order'. This nematode pest can attack other species of crop plants besides beet, and the Order prohibits the growing of any susceptible crop until the pest population has been shown by soil sampling to have declined to a very low level. This Order has helped to confine the pest to certain areas and has greatly reduced the economic importance of the beet cyst nematode in England.

These examples and many others which could be cited from numerous regions show that carefully conceived and strictly enforced legislation can play a significant part in the control of plant pests and diseases.

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Good husbandry

Appropriate cultural practices can often help to avoid or reduce damage from some pests and diseases. For example, sugar beet crops that are gappy are more prone to attack by aphids and virus yellows than are those with optimum populations. Choice of the most suitable time for sowing a crop can also influence the subsequent development of disease. In the UK early sown crops of sugar beet have been shown to be less susceptible to virus yellows than crops sown later. Conversely, early sown crops of winter wheat are often more susceptible to eyespot, a soil-borne disease caused by *Cercospora herpotrichoides*, than those which are sown later.

Crop rotation

Although this may be considered as an essential part of good husbandry, it is sufficiently important to be treated separately. Many pests and diseases, particularly those which are soil-borne, can be adequately controlled by a suitable rotation of immune and susceptible crops; indeed, in many cases this is the only practicable and economically feasible control method. Crop rotation has played an important part in the control of many soil-borne bacterial and fungus diseases, including brown leaf spot of rice (*Helminthosporium oryzae*), club root of crucifers (*Plasmodiophora brassicae*), take-all of cereals (*Gaeumannomyces (Ophiobolus) graminis*) and bacterial wilt of many crops (*Pseudomonas solanacearum*). In addition, crop rotation can give a good control of many soil-borne pests, particularly nematodes, including the cyst nematodes of sugar beet (*Heterodera schachtii*) and potato (*Globodera rostochiensis*), and species of *Meloidogyne*, some of which have a very wide host range. A rotation consisting of one susceptible crop followed by two or three resistant crops is often sufficient to prevent serious damage from these diseases. Other diseases, however, can be controlled only by unrealistically long crop rotations; the resting sporangia of the potato wart disease fungus *Synchytrium endobioticum* can remain viable for at least 10–12 years in the soil. In addition, crop rotation cannot control diseases that are spread by air-borne spores or vectors.

Clean seed or propagating material

Seeds, fruits, tubers, bulbs, corms and cuttings, if derived from infected plants, can harbour certain diseases. As far as possible, therefore, propagating material of all kinds should be taken only from disease-free plants. Where this is not possible, control measures, including the application of chemicals or heat to propagating material, can sometimes be taken to reduce the likelihood of transmission of disease. Certain seed-borne fungal diseases, e.g. bunt of wheat and covered smut of barley, in which the pathogens are carried on the seed, can be easily and inexpensively controlled by dressing the seed with an appropriate fungicide. In others, e.g. loose smut of barley, in which the pathogen is present as mycelium inside the embryo, most seed dressings are ineffective. In such diseases, heat treatment of the seed can give a good control and certain systemic fungicides have also been effective.

All organs of systemically virus-diseased plants are usually infected and, if used for vegetative propagation, will invariably give rise to diseased progeny. Although such material can often be freed from viruses by culture of meristematic tissue, sometimes in conjunction with heat treatment, such processes are laborious and expensive; they are, therefore, usually reserved for material of special value or importance from which further propagating material will be produced. Kassanis (1957) obtained plants of the potato variety King Edward freed from paracrinkle virus by meristem culture, and the new stocks derived from these virus-free plants had a greater yield than the original virus-infected stocks.

Tubers, bulbs and runners of plants can also carry fungal pathogens. For example, potato tubers may be infected with several diseases including gangrene (*Phoma exigua* var. *foveata*), dry rot (*Fusarium caeruleum*) and stem canker (*Corticium (Rhizoctonia) solani*), and such tubers may produce infected plants. Careful examination of tubers for disease symptoms and the planting of only healthy tubers will help to control these diseases.

Hygiene or sanitation

The removal of as many potential sources of infection as possible can contribute greatly to the control of plant diseases. The removal (roguing) of infected plants within a crop or the excision of diseased parts of trees are examples of good hygiene which may stop or delay the spread of infection to adjacent healthy plants. Successful roguing depends largely on the ability of the operator to recognize diseased plants, preferably at an early stage of infection. This requires training and skill and is laborious and time-consuming. Roguing is therefore generally restricted to perennial crops of high value; it is normally not economic to rogue annual crops.

Roguing has made a worth-while contribution to the control of many diseases. These include leaf roll and virus Y in potatoes in the UK, canker (caused by *Xanthomonas citri*) in citrus in the USA, phony disease of peach in the USA and banana mosaic in Honduras. These and other examples of successful disease control by roguing have been discussed in greater detail by Wheeler (1969).

There are some diseases in which roguing has been a much less successful control measure. A large-scale campaign in West Africa to cut down and burn cocoa trees which showed symptoms of swollen shoot has given only a partial control of the disease. Similarly, the spread of the Dutch elm disease has been controlled only to a small extent by destroying diseased trees, and many large-scale eradication programmes both in the USA and western Europe have been abandoned because they were ineffective. If a disease spreads rapidly, particularly if the main source of infection is outside the crop, roguing is unlikely to be worth-while.

Good hygiene also involves the control of volunteer (self-sown) crop plants, weed hosts and other potential sources of infection from the vicinity of susceptible crops. Sugar beet plants remaining in the ground after harvest can be important sources of virus yellows and downy mildew infections for nearby crops in the following year. Many common weeds, including shepherd's purse (*Capsella bursa-pastoris*), chickweed (*Stellaria media*) and groundsel (*Senecio vulgaris*), are hosts of sugar beet viruses, and infected plants can overwinter in

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mild climates to act as infection foci for the beet crop in the following spring. Mangolds and fodder beet stored over winter for animal feed in the following spring can also act as sources of infection; a decline in the amount of mangolds grown in eastern England has been associated with a decrease in the importance of virus yellows in the sugar beet crop. Beet grown for seed production can also be responsible for overwintering of beet viruses and downy mildew. The removal of seed crops from the main root-growing areas, the decrease in mangold acreage, and better control of volunteer plants and weeds, have been major factors in the control of virus yellows of sugar beet in England (Hull, 1961).

Nutrients and soil type

Many diseases can be avoided, or at least their effects reduced, by growing susceptible crops only on suitable types of soil. For example, powdery scab of potatoes (*Spongospora subterranea*), which can be particularly damaging in poorly drained, acidic soils, may often be controlled to a worth-while extent by improvement of drainage. Conversely, potatoes grown on alkaline, dry, gravelly soils are prone to attack by common scab (*Streptomyces scabies*); the addition of lime to the soil to control powdery scab may, therefore, encourage attacks of common scab. This underlines a potential weakness in trying to control diseases by purely cultural means; attempts to decrease one disease by such measures may tend to increase another.

Many diseases can be partly controlled by varying the nutritional status of the host plant. Extra applications of nitrogen to the soil can greatly increase the severity of certain diseases, for example powdery mildew and rusts of wheat and barley. Although the avoidance of heavy dressings of nitrogenous fertilizers will often help to control some diseases, high nitrogen is beneficial in controlling others such as take-all disease of cereals. High concentrations of potash and phosphate in the soil can help to control many diseases, as can application of certain trace elements. For example, the addition of small amounts of boric acid to the soil has been shown to reduce the percentage of sugar beet plants becoming infected with virus yellows (Russell, 1971). Boric acid and certain other nutrient treatments also reduce the severity of symptoms caused by several foliar diseases of cereals, including brown rust and powdery mildew of barley, and yellow rust and powdery mildew of wheat (Russell and Hudson, 1973). The extent of the effects caused by these nutrient treatments varies with different genotypes of the host species.

More information is needed concerning the effects of host plant nutrition on disease control; there seem to have been very few deliberate attempts to control diseases on a field scale by the addition of specific nutrients to the host plant.

Soil sterilization

It is not economic to sterilize the soil on a large scale against pests or diseases. However, in nurseries and glasshouses it is usual to sterilize soil either by steam treatment, so that the soil temperature exceeds 93°C for 15 – 20 minutes, or by a formaldehyde drench. These treatments give an adequate control of most soil-borne organisms.

Liquid fumigants containing dichloropropene or dichloropropane – dichloropropene mixtures have been used to sterilize soil on a field scale mainly to control nematodes, and they are also very effective against insect pests in the soil, and against soil-borne diseases. These fumigants are, however, very expensive to purchase and to apply and their use is therefore mainly restricted to sterilizing the soil for high-value cash crops.

Control of vectors

Many important virus diseases of crop plants are transmitted by animal or fungal vectors (*see* page 214), the elimination of which is usually an effective control measure. For example, insecticides have been widely used on sugar beet, much of the crop in northern Europe and the USA being treated each year to control the aphids which transmit the viruses that cause virus yellows. Leafhopper breeding grounds in the USA are sprayed with insecticide to control the hoppers, which can transmit curly top virus to sugar beet root crops. Systemic insecticides, which can be translocated from one part of a plant to another, are usually more effective in controlling insect-transmitted viruses than are contact insecticides such as DDT. Stylet-borne insect-transmitted viruses, which can be acquired during feeds of short duration by the vector from an infected plant and transmitted equally quickly to a healthy plant, are particularly difficult to control by insecticides, because the vector is usually not killed until after transmission has been successfully accomplished. On the other hand, considerable success in controlling spread of persistent insect-transmitted viruses has sometimes been achieved by using insecticides. With such viruses, long acquisition and infection feed times are necessary for successful transmission, and a quick kill of the vector is therefore not necessary to control any spread.

Control of virus vectors by chemicals is generally effective only if they are applied at the most suitable time; for example, applications after a susceptible crop has been invaded by viruliferous vectors are unlikely to give a good control. If the chemicals are applied too soon, however, they may not be present in sufficient concentration when the vectors eventually arrive on the crop. Chemicals are often expensive to buy and to apply; many are toxic to mammals and to beneficial organisms, and they can pollute the environment.

Preventive chemicals

These are applied when an attack of a pest or disease is expected, but before the attack actually starts. Sprays of copper or appropriate organic fungicides, such as dithiocarbamates or organo-tin compounds, have been applied to potato plants when warm and humid weather conditions favour the spread of the fungus causing late blight, and have been very effective in reducing the damage caused by this fungus. Similarly, barley seed in the UK is often treated with dressings of ethirimol or other suitable fungicides to protect seedlings from an early attack of powdery mildew (*Erysiphe graminis*). This is considered to be an economically worth-while insurance against severe losses from powdery mildew on susceptible varieties, even though it is often far from certain that an attack will occur. Another example is the treatment of sugar beet seed with dressings of aldicarb or other systemic insecticides to protect seedlings from early attacks

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by soil-inhabiting pests and aphids. However, these insecticides become diluted as the plants grow, so that they are less effective with outbreaks of pests that occur during the late spring or early summer.

DIRECT CONTROL MEASURES

These are therapeutic measures which can be taken to decrease the economic impact of pest and disease attack after an attack has started. Plants can be freed from most diseases in several ways, some of which are too expensive or laborious to use on a field scale. However, plants can often be cured even of systemic infection by appropriate treatments such as heat, or meristem culture, such as those used to obtain virus-free propagating material. Chemotherapy has been employed extensively to free plants from fungi, bacteria, mycoplasmas and animal pests. Some chemical treatments protect against potential infection rather than cure existing infections but, as it is often very difficult to dissociate these two activities, the protective and therapeutic effects of chemicals will be considered together here.

Chemicals for pest and disease control can be divided into those which are systemic (translocated within the plant either within an organ or between organs) and those which remain at or near the site of application. Many systemic chemicals can be applied to the seed or the soil, as granules or a dust, spray or drench, so that they are absorbed by the roots and translocated to the site of action; they are usually also translocated from one aerial part of a treated plant to another, although some are distributed only within a treated organ. Non-systemic chemicals must always be applied to those sites where control of a parasite is required.

Until the 1940s, chemical control of pests and diseases was almost entirely restricted to relatively uncomplicated, non-systemic compounds. For example, simple compounds of copper, sulphur and mercury were widely used as fungicides, while nicotine, derris and pyrethrum were used as insecticides. During the last three decades many new fungicides and insecticides have been developed. The former include non-systemic dithiocarbamates (organic sulphur compounds), such as captan and dinocap, and systemic fungicides including several compounds based on methylpyrimidine or benzimidazole. Insecticides include DDT and benzene hexachloride, which are contact insecticides, and many organophosphate or carbamate compounds, which are systemic. Antibiotic by-products of the metabolism of some micro-organisms, including penicillin and streptomycin, have been effective against some plant pathogens, particularly bacteria. Tetracycline antibiotics are particularly effective against mycoplasmas. Most pesticides are marketed under several different trade names.

There are many serious disadvantages in relying too heavily on applications of chemicals for pest and disease control. The timing of the applications is often crucial and, as treatment can be expensive, the number of applications must be kept to a minimum. Some chemicals used in plant protection are extremely toxic to mammals; others disturb the ecological balance of non-pathogenic organisms in treated crops; many are stable compounds which pollute the environment, possibly with long-term unforeseen detrimental effects; and some can be severely phytotoxic to certain crop varieties under particular environmental conditions.

An additional potential disadvantage of chemical control is that pathogens and pests may become adapted to resist or tolerate certain fungicides or insecticides, thus making chemical control less effective. Populations of aphids which are resistant to organophosphorus insecticides have been identified on several crops, particularly those grown in glasshouses, in many parts of the world. Some fungal pathogens have similarly become adapted to resist the effects of certain fungicides (Georgopoulos and Zaracovitis, 1967). Among well-documented examples of such adaptations are tolerance to benomyl in *Cercospora beticola* and *Botrytis cinerea*, tolerance to mercury in *Helminthosporium* spp. and tolerance to ethirimol in *Erysiphe graminis* (Wolfe, 1972; Dovas, Sylakakis and Georgopoulos, 1976). It seems probable, therefore, that where a particular chemical is widely used in crop protection for a long period, there is a danger that new variants of the parasites concerned may arise and that these parasites will no longer be effectively controlled by that chemical. This is a situation which closely parallels that experienced with some kinds of inherited resistance to certain pests and diseases (see page 22). On the other hand, some chemicals, for example copper and sulphur compounds, have continued to give an excellent control of many fungal diseases after widespread use over a period of many decades. Nevertheless, because of the expense of chemicals, the time and labour involved in applying them, the potential danger of polluting the environment, and the possibility of new pesticide-resistant variants of the parasites concerned, it is unwise to rely too heavily on chemical control. Pesticides should be used only when there is no satisfactory alternative, or to supplement other methods of control. Reliance on chemical control as the main defence against pests and diseases seems to the author to be an admission of defeat.

BIOLOGICAL CONTROL

Biological control involves the use of living organisms to control pests and diseases. In its widest sense it includes control by hyperparasites (parasites which are parasitic on other parasites), predators and resistant plants.

Hyperparasites and predators

One of the most successful methods of biological control has involved the introduction of natural enemies of particular insect and mite pests into an area where they did not previously exist (Van Den Bosch, 1971). This method has resulted in the complete control of the cottony cushion scale insect on citrus in California.

Another commonly used method has been the mass production of parasites or predators in the laboratory for release in areas where crops are threatened by a pest which they can control. These beneficial organisms include coccinellids (ladybirds) to control aphids, and insects which lay eggs and develop inside the bodies of pests. Predatory mites have successfully controlled the red spider mite (*Tetranychus urticae*) in glasshouse crops (Hussey, 1965). Other methods include the release of males of insect pests after they have been sterilized by irradiation or by chemicals; the introduction of lethal genes into pest populations; and the multiplication and release of bacterial or viral pathogens of pests.

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Although biological control using hyperparasites and predators has given very satisfactory results in controlling some pests, it has been unsuccessful in controlling many others. This form of control is often slow and subject to environmental influence: nevertheless, these methods are usually inexpensive and generally have no adverse effects. In addition, there has been very little, if any, development of resistance by pests to hyperparasites or predators.

Very little work has been carried out on the control of plant diseases by means of hyperparasites. Many plant pathogens can be attacked by their specific hyperparasites, however, and further work on this topic seems to be justified. Although it is unlikely that this form of biological control will ever supplant other control measures, it may help to augment their effectiveness. In sugar beet it has been shown that a combination of aphid-resistant varieties and natural biological control of aphids by parasites and predators gives a much more effective control of aphids in the field than either method alone (Lowe, 1974).

Resistant varieties

The use of resistant varieties is probably the cheapest and most effective method of combating diseases and pests. There are, however, many difficulties in breeding resistant varieties and although there have been successes, there have also been failures. Examples of these successes and failures are given in later chapters.

Provided that inherited resistance is not associated with undesirable characteristics, such as low yield or poor quality, it is as cheap for a farmer to grow a resistant variety as one which is susceptible. However, the advantages of many resistant varieties have been short-lived because new types of pathogens or pests have arisen, or increased in frequency, so that the new plant varieties have succumbed to the parasites which they had been bred to resist. This situation is commonly described as a 'breakdown of resistance' although it is, more correctly, a 'breakdown of control'. The new varieties have not lost their resistance, they have succumbed to a form of pest or pathogen to which they were never resistant. The rate at which the 'breakdown' occurs seems to depend, not only on the variability of the parasite, but also on the host species and the type or mechanism of resistance. Although much emphasis has been given to the failure of resistance to control pests and diseases, because of parasite variability, there are many examples of resistance which have continued to give a good control in widespread and protracted agricultural use. In such cases, many of which are considered in later chapters, the variability of the parasites concerned has not been important, for reasons which will be discussed.

It is not always necessary or desirable to breed for a very high level of resistance. Incomplete resistance has often given an adequate level of control in the field, particularly when such resistance has been supported by other control measures.

Combinations of Control Methods

The use of control methods in combination is called integrated control. Such control is usually better than any single method alone. The control of virus yellows of sugar beet in the UK is an excellent example of integrated control.

The economic importance of this disease, which is caused by a complex of aphid-transmitted viruses, has been greatly reduced by a programme combining strict control of hygiene, good husbandry, control of the aphid vectors by insecticides, and the cultivation of varieties which are not very susceptible either to attack by the aphid vectors or to the viruses which they transmit (Hull, 1961). The importance of integrated control measures, particularly those involving the use of resistant varieties, is considered in more detail later.

The Challenge Facing the Plant Breeder

The plant breeder, in consultation with plant pathologists or entomologists, must first decide what should be the priorities in breeding for resistance to pests and diseases. It is not just a question of concentrating on those pests and diseases which cause, or are capable of causing, the greatest amount of damage to the crop concerned. For example, a potentially damaging pest or disease, which can be satisfactorily controlled at low cost by other means, does not merit a very high priority in resistance breeding programmes; preference should be given to those that can cause serious damage and for which there are no satisfactory alternative control methods.

Having decided which pest and disease problems should have the highest priorities, the plant breeder should find out as much as possible about the biology of the causal parasites and their relationship with their host plants. This information, which is necessary for the development of suitable methods of testing and selecting for resistance, will often already be available. Sometimes, however, it is necessary for a plant breeder to initiate new studies to obtain the relevant information and this can be time-consuming and expensive. Nevertheless he must know, for example, how a pest or pathogen spreads in the field before he can devise appropriate methods of artificially inoculating his plants or of inducing epidemics, so that testing can be carried out under semi-natural conditions.

Having learnt the basic biology of the parasite concerned, and having devised effective techniques for comparing the responses to inoculation of different host genotypes, it is then necessary for the breeder to find sources of resistance which can be exploited in a breeding programme. Sources of resistance to a particular parasite will often have been discovered already by other plant breeders, perhaps in another part of the world: such material will usually be made available on request. If resistant breeding material is not readily available, a wide range of breeding lines and indigenous varieties must be examined for signs of worthwhile levels of resistance. If none is found, resistance should be sought in exotic varieties or in related, interfertile species. It is always better to use resistance from indigenous material if possible because this will already be adapted to local conditions. This will facilitate the production of resistant cultivated varieties for local use.

The methods which should be used to introduce resistance into existing breeding material or cultivated varieties will depend on several factors, including the breeding system of the crop species concerned and the type of resistance involved. In a self-pollinating crop, such as wheat or barley, resistance can be introduced by crossing resistant plants with those of susceptible breeding lines or locally adapted varieties. The resistance can easily be stabilized by allowing

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self-pollination to occur in succeeding generations with selection for resistance in the F_2 and subsequent generations. The situation is rather more complicated in cross-pollinated crops. A common method is to allow F_1 plants, derived from a cross between resistant and susceptible plants, to interpollinate and for the breeder to select recurrently for resistance. This kind of selection programme is complicated by the need to select for characters other than resistance, particularly yield and quality, in each generation. This is essential to avoid the production of highly resistant varieties which are unacceptable to the grower because they do not perform well in the absence of the pest or disease concerned.

It must be emphasized that resistance to a pest or disease is only one of many characters which have to be considered by a plant breeder. Only rarely has the importance of resistance to a parasite overshadowed all other considerations. One example of such a situation was resistance to the curly top disease of sugar beet in the USA (*see* page 243). In the 1920s and 1930s this disease caused so much damage in the western USA that the crop became uneconomic with the existing susceptible varieties. A 'crash' programme of breeding for resistance to the curly top virus produced, within a few years, varieties with a considerably greater level of resistance. This resistance was gradually improved and growing sugar beet again became profitable in these areas. However, the need to breed varieties with resistance to curly top had been so urgent that the maintenance or improvement of yield and quality of the beet in the absence of disease could not be given a high priority. The first curly top-resistant varieties were therefore comparatively low-yielding and of poor quality. Only when a satisfactory level of disease resistance had been attained was it possible to concentrate on breeding varieties with better yield potential and improved quality.

This situation was exceptional, and pest and disease resistance is usually not of such overriding importance. Nevertheless, many otherwise excellent crop varieties have not been fully exploited because of their susceptibility to a pest or disease. It is important that, in breeding for resistance to one pest or disease, a plant breeder does not neglect resistance to other parasites or other agronomically important characters. As a general rule, therefore, a resistant variety must be as good as other varieties in nearly all respects in the absence of disease; if this is not the case, that variety is unlikely to be grown on a large scale, no matter how good is its resistance to a particular pest or disease.

References

The references cited in this chapter, together with those for Chapter 2, are listed in References — Part I, pages 42–44.

GENERAL PRINCIPLES AND METHODS OF BREEDING FOR RESISTANCE

Historical

Most species of crop plants evolved during the period of very primitive agriculture, at first by natural selection and then by deliberate selection, by farmers, of plants with desirable characteristics. Both types of selection favoured plants which were best fitted to survive, but deliberate selection particularly picked out those which gave the best yields of good quality produce. Both natural and deliberate selection for improved yield and quality select plants which are not exceptionally susceptible to damaging pests or diseases because very susceptible plants are eliminated by these agents. 'Land races' (as old-established local stocks of crop species are often called) are therefore not usually very susceptible to any pest or disease with which they have been in contact for many years. Until comparatively recently, probably less than one hundred years ago, a crop species generally consisted of a series of locally adapted land races, which were perpetuated either by the grower himself or by other growers for sale locally. Much of the wheat grown in the eighteenth and nineteenth centuries in England consisted of a series of local land races, collectively known as 'Squareheads'; these had tall straw and, by modern standards, gave only a low yield of grain. Nevertheless, as far as one can judge from present-day derivatives of Squareheads, these land races did not suffer badly from leaf-infecting diseases caused by fungi, such as yellow rust and powdery mildew, which can badly damage some modern varieties. A winter wheat variety, Browick, which was derived from a single plant selected by an English farmer from a field of Squareheads wheat in the 1850s has shown an exceptionally high level of adult-plant resistance to the yellow rust disease in recent field and glasshouse experiments.

It has been suggested that the resistance of varieties such as Browick and Squareheads would not give an effective control of this disease if they were grown under the high-fertility conditions which exist in developed countries today. Experimental evidence does not support this hypothesis, however; in recent field experiments at Cambridge, Browick has given an excellent control of yellow rust in high-fertility soils. In barley too, many varieties of land races that were grown in Europe or the USA several decades ago had quite a high level of resistance to powdery mildew, another potentially damaging fungus disease.

A similar situation existed in the potato crop with regard to resistance to late blight (*Phytophthora infestans*). The first European potato stocks were

extremely susceptible to this fungus disease and crops in many parts of north-west Europe were devastated by blight epidemics in 1845 and 1846. Over the next fifty years a considerable level of field resistance to blight, both in the foliage and in the tubers, was built up in potato varieties by natural selection and by the efforts of plant breeders. Many potato varieties which were bred in the nineteenth century and the early part of the twentieth century had enough resistance to late blight to prevent the development of further disastrous disease epidemics. Although this field resistance to late blight is not possessed by many of the varieties which were developed by plant breeders during the 1940s and 1950s, these varieties carry major genes (the so-called *R* genes) derived from *Solanum demissum* (a wild relative of the cultivated potato), which control race-specific resistance to late blight.

These examples show that, even when there were no planned programmes of breeding for resistance to diseases, varieties in the past often had enough resistance to pests and diseases to prevent the frequent occurrence of disastrous epidemics. Nevertheless, because these varieties were sufficiently susceptible for farmers to sustain some losses from pests and diseases in most years, one of the main tasks of plant breeders has been to attempt to improve the levels of resistance so that these losses are minimized.

A report by Biffen (1907), that resistance to yellow rust in wheat is controlled by a single recessive gene, stimulated plant breeders and geneticists to search for genes for disease resistance in wheat and other crops. This was the start of an era of scientifically based plant breeding for resistance to pests and diseases.

Resistance in Relation to Other Breeding Objectives

The avoidance of extreme susceptibility to the most important pests and diseases should be a major objective in any plant breeding programme. In many programmes, a very high level of resistance (often referred to as 'immunity') has been sought and this necessitates an approach which differs from that which seeks merely to avoid the production of extremely susceptible varieties. The latter approach usually requires only that very susceptible plants are discarded from the breeding material, whereas the former usually involves special selection programmes in which only the most resistant plants are retained. Such programmes have often constituted a large proportion of the total effort devoted to producing new varieties, probably to the detriment of improving other agronomically important characters. Resistance to pests and diseases is only one of many breeding objectives, although it is often one of the most important. However, even the most resistant varieties are unlikely to become widely grown if they are less productive and of lower quality than other varieties.

In all agricultural crops the usefulness of a new variety will depend primarily on its yielding ability and the quality of its product; improved yield and quality are therefore generally the two most important breeding objectives. Disease and pest resistance is an important complementary objective because a high degree of susceptibility will generally result in decreased yield and quality.

Several indirect factors may increase the importance of resistance to pests and diseases as a breeding objective. For example, the acceptance of a variety may be jeopardized if it is too susceptible to any disease, even to one which is

rare or sporadic. The National Institute of Agricultural Botany (NIAB) can withhold the recommendation of a variety in England if it is considered to be too susceptible to certain diseases; several high-yielding winter wheat varieties have recently been refused recommendation by the NIAB solely because they are too susceptible to yellow rust. In such circumstances, resistance to yellow rust becomes a very important breeding objective, perhaps more so than is justified by its present or potential economic importance. Nevertheless, the growing of very susceptible varieties on a large scale is to be discouraged because this might endanger other varieties by increasing the inoculum potential of the parasite concerned. In most circumstances, however, resistance to pests and diseases should assume no greater importance in breeding programmes than many other 'secondary' breeding objectives, which in winter wheat, for example, would include early maturity, winter hardiness and the height and strength of the straw. In some crops, plant breeders may have placed too much emphasis on breeding for improved pest and disease resistance.

Terminology and Some Theoretical Concepts

A **parasite** is an organism or virus which lives upon or within another living organism at whose expense it obtains some advantage without compensation to the host. In this book, the term **pest** is applied to any animal or higher plant which parasitizes crop plants (e.g. nematodes, insects, birds and parasitic flowering plants). The term **disease** will be restricted to disorders of crop plants that are caused by pathogens, i.e. disease-causing micro-organisms such as bacteria, fungi, mycoplasmas and viruses. This distinction between pests and pathogens conforms to the terminology usually employed by plant pathologists and applied zoologists.

Most plants are **immune** (completely resistant) to most parasites. A plant is **susceptible** to any pest or pathogen which can attack it, but there are different degrees of susceptibility. Immunity is absolute and there is no disease or pest attack whatsoever. The term **resistance** may be qualified by such words as 'high', 'intermediate' or 'low' because there may be a complete gradation from extreme resistance to extreme susceptibility. Although the effects of extreme resistance and true immunity are often very similar, leading to an absence of disease or pest attack in the field, the underlying principles and mechanisms that are involved are completely different. A resistant plant can usually be rendered more susceptible by different treatments – an immune plant is never susceptible under any circumstances.

Immunity will rarely be mentioned because, although immunity is the rule rather than the exception, this book is concerned only with parasites to which crop plants are susceptible; immune plants are not attacked by these parasites and plant breeders do not need to produce varieties which are resistant to them. It is of little practical interest to farmers, plant pathologists and plant breeders, for example, that sugar beet is immune to the powdery mildew disease of barley, or that powdery mildew of sugar beet does not affect barley. What concerns us is not immunity but differences in the **degree** or **level** of resistance to pests and diseases which attack crop plants. If an individual plant of a crop species is not immune to a particular parasite, it is unlikely that any other

individuals in that species will be immune. However, there is every probability that certain individuals in the species will be more resistant (or more susceptible) to that parasite than other individuals. It is with the selection and exploitation of inherited differences in the degree of resistance that this book is mainly concerned.

The term **resistance** has been used in many different senses and it is therefore important to define how it is used in this book. **Resistance is any inherited characteristic of a host plant which lessens the effects of parasitism.** In other words, resistant plants are less damaged by parasites than are susceptible plants. This is a broader definition of resistance than that suggested by Robinson (1969) who defines resistance as the ability of the host to hinder a pathogen or disease-causing agent. This broader definition of resistance is concerned with reduction of the severity of a disease or pest attack and does not necessarily imply any direct inhibitory effect of the host plant on the development or activities of the parasite concerned. All forms of escape or avoidance of disease or pest attack that render a plant less likely to be parasitized, are included in this definition. Included in disease escape would be the forms of resistance to sugar beet virus yellows that depend on resistance to insect vectors, which transmit the viruses from plant to plant (see page 338). Certain factors can reduce the deposition of spores of fungal pathogens on the leaf surfaces of some sugar beet, wheat and barley varieties, which thereby suffer fewer infections. Similarly, some varieties of apple with late-breaking buds avoid serious infestations by several insect pests that attack early in the growing season (Briggs and Alston, 1967). Disease escape attributable to inherited factors of the host plant can thus be important in pest and disease control and it seems illogical to exclude them from a consideration of breeding for resistance, as would become necessary if a narrower definition of resistance were to be adopted.

It would also be wrong to exclude **tolerance** from any consideration of resistance. A plant which is attacked to the same degree as other plants, but which suffers less damage (in terms of yield or quality) as a result of the attack, is said to be **tolerant** (Robinson, 1969). The development of a parasite is not necessarily impeded in any way in a tolerant plant (Schafer, 1971). Indeed, some sugar beet varieties that are tolerant to beet yellows virus contain even higher concentrations of virus than more sensitive (intolerant) plants. In spite of many theoretical disadvantages of breeding for tolerance to diseases, great economic benefits have resulted from the widespread use of virus-tolerant varieties in more than twenty crop species (Posnette, 1969). Tolerance to attack by many insect pests has also been exploited successfully.

Several different types of resistance have been recognized and these have been classified in different ways. For example, the mode of inheritance has been used to distinguish three main kinds of resistance, **monogenic**, **oligogenic** and **polygenic**, in which resistance is controlled by one, a few, or many genes, respectively. Where one or more resistance genes of large effect can easily be identified, they are said to be **major genes**; **minor genes** are those which individually have only a small effect on the expression of resistance.

Van Der Plank (1963) suggested that two main types of resistance can be distinguished in epidemiological terms: **horizontal resistance**, which is effective against all genetic variants of a particular parasite, and **vertical resistance**, which is effective against certain variants only. The terms **horizontal** and **non-race-specific resistance** are synonymous but must be used cautiously because it is

never possible to test varieties or breeding material against all possible genetic variants of a parasite. The resistance of many crop varieties has been described as non-race-specific until they have been attacked by **resistance-breaking** variants of the parasite concerned! For this reason, Johnson and Law (1975) have proposed that the term **durable** is more appropriate than either non-race-specific or horizontal to describe examples of long-lasting resistance. Durability does not imply that resistance is effective against all variants of a parasite, but merely that the resistance has given an effective control for many years and is still effective. The term **transient** will be applied to resistance which has given an effective control of a parasite for only a short period because of the development of resistance-breaking variants of that parasite.

Field resistance is a term commonly applied to resistance which gives an effective control of a parasite under natural conditions in the field, but which is difficult to detect or to characterize in laboratory or glasshouse tests. Although field resistance is often polygenically controlled and has often been durable, these are not essential criteria of field resistance. Where field conditions can be satisfactorily simulated in the glasshouse or laboratory, field resistance can often be detected under artificial conditions; hence, in this respect, the use of the word 'field' is not strictly accurate. However, the term field resistance is very widely used and it is a valuable general term to describe complex kinds of resistance that give a partial control of a parasite under natural field conditions. The terms **general** or **generalized resistance** have been used in many contexts, but usually as vague alternatives to non-race-specific (horizontal) resistance or to field resistance, and they have therefore been avoided as much as possible in this book.

Different types of resistance have also been classified according to the different kinds of resistance mechanisms that are involved. For example, **active** (or perhaps more correctly **reactive** or **responsive resistance**) concerns resistance reactions of the host plant which occur in response to attack by a parasite, for example the formation of phytoalexins (antifungal compounds) by some host plants in response to inoculation with certain pathogens. **Passive resistance** involves resistance mechanisms of the host which are already present before the attack; for instance, resistance to a fungal pathogen may be attributable to a thick cuticle, which prevents infection or impedes sporulation of the fungus, or to a preformed antibiotic compound which is present in healthy plants.

Special terms have also been used to describe resistance which is expressed at different growth stages of the host plant. **Seedling resistance** can be identified in very young plants although the resistance may persist and be displayed by older plants; conversely **adult** or **mature plant resistance** is difficult or impossible to identify in seedlings. Seedling resistance is often used as a synonym of major-gene, race-specific resistance but this is not justified because adult-plant resistance can also be controlled by major genes and is often highly race-specific.

Qualitative resistance is a useful general term to describe resistance in which the frequency distribution of resistant and susceptible plants in a population is discontinuous. With this type of resistance it is easy to classify individual plants either as resistant or susceptible or into well-defined classes of susceptibility. In **quantitative resistance** on the other hand, there is a continuous gradation between resistance and susceptibility within a population of plants and there is therefore no clear-cut distinction between resistant and susceptible

plants — they form a single spectrum consisting of different degrees of resistance and susceptibility.

Attempts have also been made to define resistance in terms of the anatomical, morphological, physiological or biochemical features of the host plant which are responsible for its expression: these features are often referred to as **resistance mechanisms**. For example, several different types of components of resistance to downy mildew (*Peronospora farinosa*) have been recognized in sugar beet (Russell, 1972). These include (1) a tendency to escape infection (**disease escape**), associated with a low percentage of spore germination on the leaf surface (Russell and Evans, 1968); (2) **poor establishment of the fungus** in the host plant; (3) **slow growth of the fungus** in the host plant, either because of antibiotic substances in the host tissues or from lack of nutrients, which are essential for rapid growth of the pathogen; (4) **resistance to sporulation**, causing delayed, sparse sporulation; (5) **tolerance**, in which infected plants show little stunting or distortion of the leaves (Russell, 1972).

Similar components of resistance, including disease escape, poor establishment of infection, restricted growth of the pathogen, and delayed or sparse sporulation, have been recognized in many other fungal diseases of several crops, for example yellow rust of wheat (Russell, 1976). Individual plants may exhibit one or several resistance components, the resistance of plants with more than one type of resistance presumably being more stable or durable than resistance which is based on a single resistance component.

Each main component of resistance to a disease may consist of several, possibly independent, subcomponents. For example, disease escape in the case of leaf pathogens (such as *Erysiphe graminis* and *Puccinia striiformis*) may involve sparse deposition of spores on the leaves, a low percentage of spore germination and a thick cuticle which may be difficult for the pathogen to penetrate (see page 56). **Hypersensitivity**, defined by Stakham (1954) as the rapid death of infected cells which restricts the spread of obligate pathogens, is now thought to involve several different mechanisms (Ellingboe, 1972). Poor growth of a pathogen on or within a host plant may be caused by one or more of several different resistance mechanisms, including antibiosis (which may involve the production of inhibitory compounds by the host plant in response to infection), the presence of mechanical barriers (such as cell walls which cannot easily be degraded by the pathogen's enzymes) or the absence or insufficiency of nutrients or other essential compounds.

Several different types of resistance to animal pests have been recognized. Painter (1951) distinguished three main types of resistance to insects: **non-preference**, **antibiosis** and **tolerance**. As the name implies, insects are more reluctant to colonize non-preferred plants. The main effects of antibiosis are to retard the growth of individual insect pests and to decrease their rate of reproduction. Tolerant plants can better withstand the effects of pest attack than can sensitive plants, whether these effects are caused by direct feeding damage or by the injection of toxins into the host's tissues by the pest. All three types of resistance (non-preference, antibiosis and tolerance) have been demonstrated in many crop species, with several different types of animal pests including insects, mites, slugs, nematodes and birds. **Pest avoidance**, which is equivalent to disease escape, is an important additional type of resistance and is likely to be non-race-specific.

Parasites, whether they are fungi, viruses, bacteria, insects, nematodes or

vertebrates, show inherited variability, which must always be considered in breeding for resistance because forms may evolve or increase that can attack previously resistant varieties. Many terms have been used to describe different phenotypes and genotypes in parasite populations, including **physiologic race**, **strain**, **biotype**, **pathotype** and **variant**. The latter is a very useful general term for describing different forms of a parasite. The term **physiologic race** has been widely used for many years, particularly in connection with fungal pathogens and with race-specific (vertical) types of resistance. **Pathotype** is a more correct term but is less widely used than physiologic race to describe individuals which have a particular pathogenicity in common (Robinson, 1969). Although the term **biotype** has been used frequently to describe variants of animal pests, most nematologists prefer to use **pathotype**. **Strain** is the most common term for variants of plant pathogenic viruses.

Because different terms have been used to describe variants of different plant parasites, it is difficult to standardize the terminology, particularly as no one term is used universally. Accordingly, in this book the terminology used in connection with individual pests or pathogens is that which is most widely used for the particular parasite concerned. For example, variants of fungal pathogens which differ in virulence will be termed 'races' or 'physiologic races', variants of insect pests will generally be referred to as 'biotypes' and the term 'pathotype' will be used for variants of nematode pests. Variants of viruses will be referred to as 'strains'.

Variants of a parasite which together constitute a group of similar virulence (i.e. what is usually called a race, pathotype, strain or biotype) are not necessarily similar in characteristics other than virulence. For example, a particular physiologic race of *Puccinia graminis* (the stem rust fungus of wheat) is able to infect particular host genotypes which other races cannot attack. However, individuals comprising this race may differ from each other in many other respects, such as vigour, spore colour and temperature sensitivity. A physiologic race is therefore not a homogeneous entity and may comprise widely differing individuals, their only common factor being an inherited ability to attack particular genotypes of the host plant.

To most bacteriologists and virologists, **virulent** strains of a pathogen are those which cause more severe symptoms of disease than those which are **avirulent**. Much confusion has arisen from the use of the term 'virulent' by mycologists in a quite different sense; a **virulent** physiologic race of a **fungal** pathogen is one which carries 'virulence' genes which enable it to attack a particular host genotype; an **avirulent** race cannot attack this genotype. An **aggressive** or **vigorous** race of a fungal pathogen is equivalent in many ways to a virulent strain of a virus in that they both cause severe disease on all those host genotypes which they are able to attack. Virologists also commonly refer to a 'severe' or 'mild' virus strain, these terms in fact describing the severity of the disease which is caused; 'virulent' and 'avirulent' are more correct for this purpose. It should be realized, however, that a strain of a bacterial pathogen or virus which is virulent on plants of one host species is not necessarily also virulent on those of other host species.

Although attempts have been made recently to standardize the meaning and use of terms employed in plant pathology (e.g. Robinson, 1969; Federation of British Plant Pathologists, 1973; Robinson, 1976), there is still considerable confusion and disagreement. This is inevitable until more is known about the

nature of resistance to parasites and about the genetics of host–parasite interactions. The ways in which particular terms are used in this book have been defined in this section; although it is unrealistic to expect all breeders, geneticists and pathologists to agree with every definition, readers will at least be able to understand the ways in which the terms have been used.

The Importance of Parasite Variability to the Plant Breeder

The great, and often unexpected, ability of many pathogens or pests to become adapted so that variants can attack previously resistant varieties has been one of the main problems encountered by plant breeders. There are many well-documented examples of the resistance of cultivated varieties ‘breaking down’ because of the occurrence of variants of parasites which are able to overcome the resistance. For example, several wheat varieties, including Kanrad, Kota and Ceres, showed good resistance to stem rust when they were first cultivated in the USA, but they soon succumbed to new physiologic races of *Puccinia graminis*, the causal fungus; as a result, millions of acres of wheat in the USA were devastated by stem rust (see page 90). In Europe, during the past twenty years a succession of high-yielding wheat varieties that initially showed good resistance to yellow rust (*Puccinia striiformis*) were severely attacked by virulent physiologic races of the pathogen soon after they were released. Potato varieties that at first were highly resistant to the potato blight fungus (*Phytophthora infestans*) have been shown to be susceptible to certain physiological races of the fungus, which become widespread wherever these varieties are grown (see page 117).

Strains of viruses and insect biotypes that are able to attack resistant varieties have generally been less of a problem to plant breeders than have physiologic races of fungal pathogens. However, breakdown of resistance has occurred with some virus diseases, for example tobacco mosaic in tomato (see page 257). Variants of some insect pests have also been found that are able to attack previously resistant varieties. Several biotypes of *Mayetiola destructor* (the Hessian fly) can attack wheat varieties that are resistant to other biotypes (see page 326). Resistance to some nematode pests can also be very race-specific, and many pathotypes of the potato cyst nematode (*Globodera* spp.) have been identified (see page 358).

Most resistance-breaking variants of pathogens and pests do not arise *de novo* because resistant varieties are grown. They are usually present in very low proportions in parasite populations and become more frequent and more widely distributed only as a result of strong selection pressure in their favour, when varieties are grown which are resistant to some variants but susceptible to others. For example, variants of *Erysiphe graminis* f.sp. *hordei* (the powdery mildew fungus that infects barley) with virulence genes corresponding to specific resistance genes in barley have been identified in *E. graminis* populations in areas where barleys with these resistance genes have never been grown. Similarly, insecticide-resistant variants of aphids and fungicide-tolerant fungi have been found in low frequencies in areas where pesticides and fungicides have never been used.

In breeding for resistance to pests and diseases, resistance-breaking variants of pests and pathogens have not always been very damaging in practice. Resistance has often been durable and sometimes apparently non-race-specific,

and, in other cases, resistance has given a worth-while control for many years in most areas, even when resistance-breaking variants are known to be present. For example, several rice varieties, including Tatep, have shown a high level of resistance to the rice blast fungus (*Piricularia oryzae*) in many parts of the world, although they have been shown experimentally to be very susceptible to certain races of the pathogen (see page 111). On the other hand there have been many instances in which the occurrence of resistance-breaking variants has caused severe damage and has led to the premature withdrawal of high-yielding varieties, as in rust diseases of wheat (see pages 83–103) and powdery mildew of barley (see page 104).

Why has the variability and adaptability of parasites been a serious problem in overcoming resistance with some pests and diseases but not with others? The following ten queries seem to be relevant in attempting to answer these questions:

- (1) Are some parasites capable of greater genetic variability than others?
- (2) Is the breeding system of a parasite (e.g. whether it is autoecious or heteroecious, monokaryotic or dikaryotic, sexual or asexual) important in determining its ability to produce resistance-breaking variants?
- (3) Is durable resistance achieved more easily against parasites that are soil- or seed-borne than against air-borne parasites?
- (4) Are problems with resistance-breaking variants more likely to occur with obligate parasites (i.e. those that can live only as parasites) than with facultative parasites (i.e. those which can exist saprophytically) – or with parasites with narrow rather than wide host ranges?
- (5) Is it easier to breed varieties with durable resistance in crop species with particular kinds of breeding system (i.e. self-pollinators or cross-pollinators, annuals or perennials)?
- (6) Is there a relationship between durability of resistance and any particular kind of inheritance of resistance, such as control by major genes, minor genes, polygenes or extra-chromosomal genes?
- (7) Does the ploidy level of the host affect the chances of achieving durable resistance?
- (8) Is resistance derived from cultivars likely to be more durable than resistance from closely related wild forms or from other species?
- (9) Are certain types of host-plant resistance (resistance mechanisms) more likely than others to be overcome by new variants of parasites?
- (10) Are combinations of different resistance mechanisms to a parasite generally more durable than single mechanisms?

One of the purposes of this book is to examine evidence from breeding programmes for resistance to a wide range of pests and diseases in some of the major agricultural crops in an attempt to answer some of these queries. The evidence will be summarized and discussed in Chapter 13.

Sources of Resistance

The first requirement of any programme of breeding for resistance must be to find a usable source of resistance. Such sources may be present in existing or old varieties, in wild forms of the same species, in closely related species, or even in

different genera. The first of these possible sources is the most useful because there should be no problems of infertility such as occur in interspecific hybrids, and agronomically undesirable characters derived from wild plants or another species do not have to be bred out. The second type of source (wild relatives of the same species) can sometimes present very great difficulties. An example is the attempt to use in breeding the wild sea beet (*Beta vulgaris* s.sp. *maritima*) which is closely related to cultivated sugar beet and with which it is completely interfertile. Although some populations of wild sea beet are more resistant than sugar beet to several diseases, including virus yellows and *Cercospora* leaf spot, it has proved almost impossible to transfer resistance from it without also transferring a large number of undesirable characteristics of the wild parent. Furthermore, resistance to each of these diseases is apparently controlled by several genes so that much of the resistance would probably be lost in the many backcross generations necessary to eliminate the undesirable features.

Simply inherited resistance to several diseases has been successfully transferred by crossing susceptible varieties with resistant plants of another species. Many different techniques have been used in such interspecific crosses. Occasionally, different species are interfertile, and normal hybridization techniques can be employed to produce fertile F_1 plants from the interspecific cross; in these cases, the two species generally have similar chromosome numbers and chromosome homology. Crosses between American Upland cotton (*Gossypium hirsutum*) and Egyptian cotton (*Gossypium barbadense*) are fertile, and hybridization between them has been used in breeding for resistance to bacterial blight (*Xanthomonas malvacearum*) in cotton (see page 179).

In other cases it may be possible to obtain amphidiploid hybrids from interspecific crosses. Fertile amphidiploids have been obtained, for example, from interspecific crosses in many genera including *Triticum* (wheat), *Gossypium* (cotton) and *Nicotiana* (tobacco). This will often involve doubling the number of chromosomes in the sterile F_1 plants by the use of colchicine. Resistance genes can sometimes be introduced from one species to another by alien chromosome substitution or addition. This procedure in wheat and its implications in breeding for resistance have been described by Riley (1968). Although this method of chromosome substitution is of considerable interest and potential, few resistant varieties have been produced so far by its use.

Inheritance of Resistance

Although some knowledge about the genetics of resistance to a particular pest or disease is helpful in any programme of breeding for resistance, it is not necessary for the plant breeder to understand exactly how resistance is inherited before a successful breeding programme can be carried out. Indeed, details of the genetics of resistance are known in only a few cases. It is, however, useful to know whether resistance is dominant or recessive, whether it is controlled by one gene (monogenically), a few (oligogenically), or many genes (polygenically) and whether cytoplasmic (extrachromosomal) inheritance is involved.

Variation in resistance to a particular parasite may be expressed continuously or discontinuously in a segregating population, depending on the number of resistance genes that are involved. With continuous variation there is a complete gradation between susceptibility and resistance, and many resistance genes are

involved; with discontinuous variation, individuals fall into well-defined classes of resistance or susceptibility, and resistance is controlled either monogenically or oligogenically.

Two main types of resistance to downy mildew of sugar beet have been recognized (see page 129). The first and most common type involves several apparently independent resistance mechanisms and is controlled by several genes, the resistance varying continuously in a segregating population. A second type is based on a hypersensitive response of the epidermal cells to infection and is controlled by a single dominant major gene; the F_1 is resistant and a ratio of three resistant to one susceptible is found in the F_2 generation. The genetics of resistance to pests and diseases is, however, seldom as simple as this and resistance to powdery mildew in barley will serve as an example of a more complex genetic situation.

In barley there are at least seven loci with at least 17 different alleles which condition resistance to powdery mildew, caused by *Erysiphe graminis* f.sp. *hordei* (Wolfe, 1972). Eleven of these alleles are at, or near, the *Ml-a* locus, which is therefore termed a **multiple allelic locus**, and which is situated on chromosome 5 (Moseman and Jørgensen, 1971). Different alleles at the *Ml-a* locus probably control similar or identical resistance mechanisms, although corresponding virulence genes in the pathogen, which can overcome the resistance, can be allelic or non-allelic (see page 107). Most of the types of resistance to barley mildew that have so far been characterized are controlled by single genes, and many plant breeders have combined several of these non-allelic resistance genes into their varieties in the hope that the resistance would thereby be made more durable. These efforts have, however, usually been in vain; many recently produced resistant barley varieties have been severely attacked, soon after their release, by resistance-breaking races of *E. graminis* f.sp. *hordei*, and this has sometimes occurred even while the varieties were being multiplied before their release to the farmer.

Many of the loci which control resistance to powdery mildew in barley are **linked** and are inherited as a group. Linkage favours the retention of existing combinations of these resistance genes and it is not surprising, therefore, that genes for resistance to mildew are often present in combination, even when no special effort has been made to combine them. However, crossing-over can separate these resistance genes and they can therefore readily be obtained individually in a breeding programme if this is required.

Resistance genes may interact, the interactions being **complementary** where two or more non-allelic resistance genes are required to confer resistance; alternatively, a resistance gene may require the presence of another gene before it can be fully expressed (**modifying action**). One gene can also **mask** the action of another. Effects of resistance genes may be **additive** as, for example, when the expression of resistance is increased in the presence of two or more different resistance genes; alternatively, one may be dominant over another, non-allelic, gene (**epistasis**). Different genes can control the same resistance mechanism, the presence of any one of these **duplicate** genes conferring the same level of resistance as any combination of the others. A knowledge of the interactions between resistance genes can sometimes help the plant breeder to conduct his programme of breeding for resistance less empirically and therefore probably more efficiently. For example, even where resistance genes can be duplicated there is no point in doing so because they will not increase the expression of

resistance. However, where resistance genes are known to be additive in their effects, it is probably worth while to try to combine several resistance genes in a single variety to enhance the expression of resistance.

Methods of Testing for Resistance

In breeding for resistance to a disease or pest, plant populations must be exposed to the parasite in such a way that resistant and susceptible plants can readily be distinguished from each other. Every plant should be exposed as far as possible to the same level of attack as any other. Selection for resistance to some pests and diseases can be carried out very effectively in natural field epidemics. For example, powdery mildew of barley is so ubiquitous in the UK that selection and testing for resistance can be carried out in most years under natural infection conditions in the field without any special provision for encouraging infection. With many other pests and diseases, suitable sites have to be chosen carefully in order to obtain good conditions for selecting for resistance under natural conditions. Selection of sugar beet for resistance to the curly top disease in the USA is usually carried out in fields adjacent to the foothills that are the main breeding grounds for the leafhopper vector of the virus. In the spring, viruliferous leafhoppers move from virus-infected weed hosts in their breeding grounds to the sugar beet plots, where they multiply and spread the curly top virus within the breeding material. An early and uniform curly top infection can be obtained in this way, and this enables plant breeders to differentiate between susceptible and resistant plants. In the UK, potatoes are selected for resistance to aphid-transmitted virus diseases in East Anglia, where virus sources are often common and aphid vectors are usually numerous. The Toluca Valley in Mexico is particularly suitable for testing potatoes for resistance to late blight (*Phytophthora infestans*), both because the climate is very suitable for the spread of the fungus, and because many races of the fungus are present. However, natural epidemics of most pests and diseases do not occur every year, even at the most suitable sites, and the breeder often has to start artificial epidemics so that he can be assured of suitable selection conditions.

A common method of encouraging epidemics is to interplant susceptible genotypes, which are often artificially inoculated, between the plants to be tested. These susceptible plants act as foci of infection from which the pest or disease can spread. For example, in testing and selecting wheat lines for resistance to yellow rust (*Puccinia striiformis*), rows of 'infectors' or 'spreader' plants of very susceptible varieties can be planted between rows or plots of breeding material. These spreader plants are inoculated with the fungus early in the growing season to ensure that there is adequate inoculum for natural spread of the disease. In selecting for resistance to downy mildew (*Peronospora farinosa*) in sugar beet in the UK, rows of inoculated plants of a susceptible sugar beet variety are planted between the rows of breeders' lines; selection tests are carried out near the west coast of Wales where, although humid conditions favour the spread of downy mildew, the plots are not a hazard to other beet crops because sugar beet is not grown in that area. The main advantage of such methods, whether using naturally occurring or artificially induced infection foci, is that the pest or pathogen spreads naturally to the experimental plants. Artificial methods of inoculation do not have this

advantage, but they are very widely employed because they are usually more reliable and easier to carry out.

Many methods have been developed for artificially inoculating plants with pests and pathogens, the most appropriate method for a particular parasite depending on how it is disseminated and its particular environmental requirements. Some general principles concerning inoculation methods for use with different groups of pests and pathogens are outlined below. More detailed descriptions of these and other methods will be given in subsequent chapters.

FUNGI

In suitable environmental conditions, spores of fungal pathogens germinate on susceptible hosts, and penetration of the host by the pathogen occurs either through natural openings (such as stomata or lenticels) or by direct penetration through the cuticle. Plants can be inoculated by applying viable spores or pieces of mycelium of the pathogen to appropriate parts of the host plants as uniformly as possible; the inoculated plants are then incubated under conditions which favour spore germination and penetration of the host plant. The uniform application of inoculum can be accomplished in various ways:

- (1) By spraying a suspension of spores or mycelium in water or light mineral oil. Inoculation with Phycomycete fungal pathogens using suspensions of conidia in water can be very successful, but spores of many other groups of fungi are killed by immersion in water. Spores of most rust and powdery mildew fungi can be effectively applied in mineral oil but not in water.
- (2) By applying drops of a spore suspension by means of a micropipette or a syringe.
- (3) By dusting plants with dry spores, using a paint brush or similar method of transfer, or using sophisticated apparatus through which spores are blown, e.g. a cyclone-head attached to an air compressor (see Figure 3.6). Spores are often mixed with talc as a carrier to obtain a more even distribution of spores on the experimental plants. Elaborate spore-settling towers have been designed to ensure uniform deposition of dry spores on test plants (see Figures 3.7 and 3.9).

The environmental conditions that are necessary for spore germination and penetration vary greatly from one fungal pathogen to another. *Erysiphe graminis* (which causes powdery mildew of cereals) can infect plants over a wide range of temperature and atmospheric humidity but others, for example *Puccinia striiformis* (the yellow rust fungus of cereals) are very exacting in their environmental requirements. Inoculation with *P. striiformis* is most successful with air temperatures below 15°C and with high relative humidities; a film of free water on the leaf surface is required for germination of the uredospores. Although elaborate methods have been developed to produce high humidity after inoculation, it is often sufficient to place inoculated plants in plastic bags or other waterproof covers and to keep them in a cool place for 24–48 hours. Temperature and humidity can usually be controlled more easily and accurately in the glass-house or growth room than in the field.

28 *General principles and methods of breeding for resistance*

In testing for resistance to seed-borne fungal diseases, plants are usually inoculated either by dusting spores on to dry seed, a method that is used in inoculating wheat grains with the bunt fungus (*Tilletia caries*), or by soaking the seed in a suspension of spores, often under vacuum. Similar methods can be used for seed-borne bacterial diseases. The inoculated seed is then sown in the field or glasshouse where the numbers of infected plants in different genotypes are recorded.

Soil-borne pathogens usually attack the host plant at, or below, the surface of the soil. Testing and selecting for resistance to these diseases is usually carried out in field experiments in soils which are known to have a high population of the pathogen concerned. If this is not possible, attacks can be induced artificially, either by introducing soil from fields where attacks have occurred, or by culturing the parasite in the laboratory and then introducing it into the soil.

BACTERIA

One of the most common methods of inoculating plants with pathogenic bacteria is to spray them with a suspension of the pathogen in water; this method has been used in breeding for resistance to bacterial blight of cotton (see page 181). If a power sprayer is used, some of the inoculum can be forced through the stomata into the intercellular air spaces of the host plant thus facilitating infection. In work with bacterial blight of rice, several other methods of inoculation have also been used, including pricking the leaves with needles that are contaminated with bacteria or immersing seedlings in a bacterial suspension. Needle-prick inoculations have given results which closely resemble those which are obtained under conditions of natural bacterial blight infection. Hypodermic syringes have also been used to force inoculum into the leaves.

VIRUSES AND MYCOPLASMA-LIKE ORGANISMS

Viruses are obligate wound parasites and cannot, therefore, penetrate the intact cuticle of a host plant or intact cell walls. Initially, they can enter only damaged cells, although in established infections virus particles can move readily from cell to cell through the plasmodesmata. For viruses, therefore, different methods of artificial inoculation to those used with fungi and bacteria, which are not wound parasites, must be employed. These methods fall into three main categories: (1) transmission by grafting; (2) mechanical inoculation; (3) transmission by vectors. All plant viruses can be transmitted through graft unions between diseased and healthy plants. Although grafting is very laborious and time-consuming, graft transmission of certain viruses is used in large-scale programmes of selecting for resistance, for example in testing for field immunity to potato virus X, which involves hypersensitivity controlled by a single gene *Nx* (see page 236). The presence of resistance gene *Nx* in a potato plant can easily be recognized by grafting on to it a scion from a susceptible variety which is infected with potato virus X: plants with the gene *Nx* show a top-necrotic reaction two to four weeks later.

Many viruses can be transmitted mechanically by rubbing leaves or other organs of a healthy plant with a suspension of virus particles. Although it is

preferable to use a purified virus preparation, successful transmission can usually be obtained with crude or clarified sap from infected plants; for this reason, this form of transmission is often referred to as 'sap inoculation'. Transmission can usually be improved if an abrasive such as Celite or carborundum powder is added to the inoculum. The inoculum can be applied in various ways, for example by stroking the plants with a virus-contaminated finger, piece of muslin or soft brush, or by a spray gun which injects inoculum deeply into the tissues of the host plant. The latter method has been used successfully to inoculate sugar beet plants with curly top virus in screening tests; previously the only known way to transmit this virus was to use leafhoppers as vectors (Mumford, 1972).

Some plant viruses are transmitted only by specific animal or fungal vectors; others can be mechanically transmitted only with great difficulty. Inoculation of vector-transmitted viruses involves first the acquisition of virus by the vector from an infected host plant and the subsequent transfer of that vector to the plant to be inoculated. The length of time needed for a vector to acquire and transmit a virus will depend on the type of virus involved. For example, *Myzus persicae* (the peach-potato aphid) is able to transmit some viruses during feeds of only a few seconds' duration, and others only with feeds lasting many hours. There is a very specific association between individual viruses and their vectors, and most vector-transmitted viruses can be transmitted only by one, or a few, species of vector. Plant breeders must know what are the vectors of the viruses with which they are working, and also the transmission characteristics of these viruses; published information about these properties for most agriculturally important plant viruses is readily available to the plant breeder (e.g. Smith, 1972; Commonwealth Mycological Institute, 1970).

Several plant viruses are now known to be transmitted by nematodes or fungi in the soil. Tests for resistance to these viruses are usually carried out in the field by growing plots of different genotypes in soils containing both virus and vector, and where the disease is prevalent. Artificial tests can be carried out by growing plants in pots containing soil from fields where the disease is common, or by inoculating potting compost with soil containing both vector and virus.

ANIMAL PESTS

Most breeding for resistance to animal pests has been concerned with insects and nematodes. In work with plant parasitic nematodes, most of which inhabit the soil, techniques similar to those used for nematode-transmitted viruses have been employed. Breeding material has generally been screened for resistance to cyst-forming nematodes (such as the potato cyst nematode and the sugar beet cyst nematode) in infested soils, either in the field or in pots; plants with roots on which few cysts develop are considered to be resistant. Resistance to root-knot nematodes and stem nematodes is usually evaluated also by growing plants in infested soil, but in these cases the amount of damage caused by the attack, rather than the number of nematodes present, is usually the criterion for selection. Laboratory tests also have been developed to test for resistance to stem nematodes; seedlings are grown between layers of moist filter paper and are each inoculated with a drop of a suspension of nematode larvae; infested plants are

subsequently scored for severity of symptoms. The nematode larvae used are obtained either from infested soil or from infested susceptible plants.

For artificial tests of resistance to insects and mites that attack the aerial parts of plants, large numbers of the pest are often reared on appropriate host plants in insectaries. A simple but effective method of inoculating plants with insect pests, in field or glasshouse tests, is to transfer a predetermined number of individuals to each plant to be tested, or to susceptible 'spreader' plants which have been planted uniformly among the experimental plants.

Requirements for Successful Inoculation

When the natural spread of a parasite is relied upon in selection and evaluation tests in the field, these must be carried out where natural attacks can be expected to occur or where the parasite will spread from artificially inoculated plants. The plant material to be tested must be arranged so that each plant stands an approximately equal chance of becoming parasitized. It is sometimes possible to encourage the natural spread of pests or pathogens in field plots; for example, certain fungal pathogens can be encouraged by using overhead irrigation to increase humidity around the plants. However, it is usually not possible to manipulate the field environment to a significant extent, and uncontrollable adverse environmental factors often seriously decrease the effectiveness of screening tests under natural conditions in the field.

Many of these problems can be avoided or overcome by artificially inoculating the experimental plants, although minor differences in pest or disease escape may be masked by this procedure. The correct quantity of high-quality inoculum must be used to achieve the most satisfactory results. Inoculum of fungal or bacterial pathogens should contain a high concentration of viable spores or cells which are in a suitable physiological state to start infection in susceptible control plants. The dose of inoculum that is used to inoculate breeding material can be a critical factor. Each plant to be tested should receive an approximately equal amount of inoculum, which should therefore be applied with considerable precision. The required standards of precision can be achieved more easily in the laboratory and glasshouse than in the field.

In both natural spread and artificial inoculation tests, it is important to set out the breeding material in plots according to accepted principles of experimentation, including replication and randomization, so that statistical analysis of the results is possible.

Even when inoculum of the highest quality has been distributed evenly throughout the experimental material, adequate levels of infection or infestation will be achieved only if the environmental conditions favour infection. For example, uredospores of *Puccinia striiformis* (the yellow rust fungus of cereals) do not germinate at temperatures above 25°C and it is therefore useless to conduct tests for resistance to yellow rust at high temperatures. The optimum temperature for germination of uredospores of *P. striiformis* and for growth of mycelium is about 11°C and the most informative results from screening tests will be obtained at this temperature. Most other rust fungi, such as *Puccinia graminis* (the wheat stem rust fungus), require higher temperatures for optimum growth and development, and selection tests for resistance to stem rust should be carried out at temperatures of 17–18°C. Different temperature regimes are

sometimes necessary at different stages of a pathogen's development to obtain the maximum symptoms. Conidia of *Erysiphe graminis* (powdery mildew of cereals) germinate best at temperatures between 5 and 9°C, whereas optimum growth of mycelium occurs at about 20°C.

Bacteria, mycoplasmas and viruses are parasites that enter the host plant passively through natural openings or through wounds, and they are generally less temperature-sensitive at inoculation time than are fungal pathogens. Mechanical transmission of some viruses will fail, however, under unfavourable environmental conditions. For example, sap transmission of beet yellows virus from sugar beet to *Claytonia perfoliata* is usually unsuccessful at temperatures above 21°C but can be accomplished without difficulty between 10 and 15°C. Efficient transmission of viruses and mycoplasmas by insect vectors will occur only if environmental conditions favour the feeding of the vector on the host plant. Very high temperatures frequently make aphid vectors of viruses so restless that they do not feed well enough for virus transmission to occur.

With many fungal and bacterial diseases it is particularly important to maintain a high level of humidity at the site of inoculation. Uredospores of many rust fungi require a high atmospheric humidity, and also a film of moisture on the leaf surface, for spore germination and penetration of the host plant to occur. Sporangia of the potato blight fungus (*Phytophthora infestans*) germinate only under such conditions; few sporangia of *P. infestans* are produced in relative humidities of less than 97 per cent. The quality of water that is used to produce films on leaves and other plant organs in artificial inoculations with fungal pathogens is also important; tap water may contain impurities which can inhibit the germination of fungal spores (Russell and Evans, 1968).

The quality and intensity of light (and the daylength) can affect inoculation with many pests and pathogens. Periods of darkness inhibit the germination of sporangia of *Phytophthora infestans*, and potato plants should therefore be inoculated in the light when they are being tested for resistance to blight. Conversely, a period of darkness after inoculation increases the susceptibility of sugar beet to *Peronospora farinosa*, the downy mildew fungus (Russell and Evans, 1972). Periods of darkness may also affect the settling behaviour of insects on their host plants and, consequently, also their ability to transmit plant viruses. The aphid *Myzus persicae* is restless, and produces fewer larvae (nymphs) on sugar beet plants that are kept in complete darkness for 48 hours either before or after infestation, than it produces on corresponding plants grown in normal daylight (Russell, 1969). Transmission of beet yellows virus by *M. persicae* is also significantly less to dark-treated plants than to those that have been grown under normal daylength conditions.

Parasitization, even of normally susceptible plants, will occur only if the host plant is in a suitably receptive physiological state. Pot-grown wheat plants, even those of genotypes which are known to be very susceptible to yellow rust under most conditions, can be difficult to infect as adult plants if they are pot-bound or growing badly because of inadequate nutrition or moisture. Similarly, plants of sugar beet, that are known normally to be very susceptible to aphids, become very resistant to the insects when pot-bound (Lowe, 1974) and when the plants are badly wilted. The presence of other diseases can also affect the efficiency of testing and selecting for resistance to pests and other diseases. Sugar beet plants infected with some strains of beet yellows virus express greater resistance to downy mildew (*Peronospora farinosa*) than healthy

plants, but infection with other strains of the same virus can increase susceptibility (Russell, 1970). Beet plants that are badly infected with powdery mildew are usually much more resistant to *Ramularia* leaf-spot and to rusts than are mildew-free plants. Similar interactions between diseases have been reported in many crops, including cucumber and *Phaseolus* beans (e.g. Yarwood, 1957). In selecting for resistance to one pest or disease care should therefore be taken to protect the breeding material from attacks by other parasites. On the other hand, in testing sugar beet for resistance to two species of aphid, *Myzus persicae* and *Aphis fabae*, it has been shown that plants can be tested simultaneously for resistance to both species, because in this case the presence of one species does not affect the behaviour of the other (Russell, 1966).

In summary, the following factors are important in obtaining a successful inoculation in tests of resistance to pests and diseases.

- (1) The inoculum must be of high quality.
- (2) The inoculum must be applied as uniformly as possible to the host plant, at the most suitable concentration to differentiate between resistant and susceptible plants.
- (3) Environmental conditions at inoculation and during any incubation period must be suitable for the satisfactory development of the parasite concerned.
- (4) The host plants that are being tested should preferably be free from attack by pests or diseases, and should be in a suitable physiological state for infection or infestation to occur.

The Assessment of Resistance

The main objective of selection tests (whether they involve artificial inoculation or are carried out under natural conditions) is to differentiate between resistant and susceptible plants. Differentiation between very resistant and very susceptible plants should be easy, provided that the inoculation has been carried out correctly and that the environmental conditions are suitable for parasitization to occur. It is usually easy to distinguish between susceptible and resistant individuals in a segregating population of inoculated plants in which resistance is controlled by one or a few major genes; variation in resistance in such cases is discontinuous and the most resistant plants are easy to identify. However, resistance is often controlled by many genes and there is then no clear-cut distinction between resistant and susceptible plants in a segregating population; differences in resistance are quantitative rather than qualitative. Where there is a continuous gradient in the expression of resistance within a host plant population, it is necessary to measure or estimate the intensity of the disease or pest attack before effective selection for resistance is possible. There are several ways in which this can be done:

- (1) The degree of attack can be estimated by giving each plant, or group of plants, a numerical score for the severity of symptoms. The following type of 0–4 scale is often used for such estimates:
 - 0 = no symptoms.
 - 1 = very mild symptoms.

2 = mild symptoms.

3 = severe symptoms.

4 = very severe symptoms.

- (2) The number of discrete infections, or of pest individuals (such as insects or nematode cysts) on a plant can be arbitrarily assessed using a similar scoring system (e.g. 0 = none, 4 = many).
- (3) The area of the plant that is affected by a parasite can also be assessed using a simple scoring system. For example, the percentage of leaf tissue that is diseased has frequently been used to compare the severity of attacks by rust fungi on cereal plants (e.g. Calpouzos *et al.*, 1976).
- (4) In scoring for resistance to rusts and certain other diseases it is often useful to classify the **infection type** or **reaction type**. In cereal rusts, several basic reaction types are recognized according to the size and type of lesions or pustules that develop on inoculated leaves; these reaction types are described in detail on page 76.

Although these scoring systems are subjective they are generally reproducible and reliable and they are therefore adequate for most selection and testing purposes.

Where more precise comparisons of resistance are required it is necessary to undertake detailed measurements. For example, yields from individual plants or families can indicate the relative severity of the effects of pest attack or disease. In other cases the severity of disease or infestation can be accurately assessed by counting the number of discrete lesions or pustules, or the number of insects or other pests, on individual plants. Alternatively, the average size of lesion or pustule or the percentage of host plant tissue that is affected can be gauged. The extent of disease caused by fungi can also be determined by measuring the amount of mycelium present in the host's tissues or by weighing the spores produced. The weights of *Puccinia striiformis* uredospores produced on different wheat genotypes have been compared very precisely, either by weighing on a microbalance or by spectrophotometry, in attempts to demonstrate small differences in the expression of resistance to yellow rust (Johnson and Bowyer, 1975). Similarly, the gain in weight by individual aphids within a given time while they are feeding on different sugar beet plants has been used as an indication of the relative resistance of the plants to these insects. Techniques for estimating the quantity of fungal chitin on, or in, individual host plants, have been developed (Ride and Drysdale, 1972) so that the relative growth rates of fungal pathogens in different host plants can be compared. Similar techniques presumably could be used to estimate the level of resistance of different plants to insect pests, by measuring the amount of insect chitin produced on a plant within a given time. However, recent work by Sharma, Fisher and Webster (1977) suggests that chitin assays are not always a reliable method for estimating fungal biomass in plant tissues.

Precise measurements are particularly useful in studying the detailed interactions between host and parasite, but are probably too complex and time-consuming for use in large-scale breeding programmes, in most of which, therefore, less precise but more rapid methods will probably continue to be used for most purposes.

Selecting for Resistance

Although it is relatively easy to select for resistance to most pests and diseases, there may be many complicating factors. For instance, certain host plant genotypes may be highly resistant to some variants of a parasite but very susceptible to others. It is therefore desirable to test host genotypes against a wide range of variants of a parasite before selection. Such tests may use individual variants separately, in known combinations, or in populations of unknown composition obtained from the field. The use of individual variants is particularly useful in obtaining information about host–parasite relationships, but is usually not practicable for large-scale breeding tests. In large field selection programmes, breeding material can be exposed to mixtures of isolates of a parasite which have been collected from several different naturally occurring field populations. In this way, breeding material can be exposed to a very wide range of naturally occurring variants of that parasite. Known mixtures of variants can be used, so that plants are tested against several variants simultaneously; this method has the added advantage that the mixture can be reproduced at will. The mixture can be maintained either as the individual variants on appropriate host genotypes or as a mixture on a range of host genotypes. An alternative approach is to grow plots of breeding material at several different sites where different variants of parasites are expected to occur. Plants that express resistance to only a few variants will be identified by either method and can then be discarded from the breeding material.

Such methods may help plant breeders to avoid selecting plants with highly race-specific resistance. However, potential resistance-breaking variants are usually present only in very small proportions in unselected field populations of a parasite, and these will become widespread only when host plant varieties with corresponding resistance genes are grown on a large scale. The use of mixtures of variants will therefore not necessarily indicate whether or not resistance is race-specific, because resistance-breaking variants are unlikely to be included in such mixtures.

The expression of resistance to a particular parasite can vary greatly at different stages of development of a plant. Also, one organ on an individual plant may be highly resistant to a parasite, whereas another organ may be very susceptible. Two winter wheat varieties, Little Joss and Nord Desprez, are almost equally susceptible to most races of *Puccinia striiformis* as seedlings, but adult plants of Little Joss are much more resistant to this pathogen than are those of Nord Desprez (Russell and Hudson, 1974). In other words, both varieties are susceptible to *P. striiformis*, but Little Joss shows adult-plant resistance. Similarly, the first seedling leaf of certain wheat genotypes reacts to yellow rust in quite a different way from that of the second and subsequent leaves (Stubbs, 1968); the first leaf is more susceptible to yellow rust than the second in some genotypes, but is more resistant than the second leaf in others. The ears of certain wheat varieties are much more susceptible to yellow rust than the leaves, whereas in other varieties the leaves are more susceptible than the ears. There is often only a poor correlation between the levels of resistance to *Xanthomonas malvacearum* shown by the leaves, stems and bolls of the same cotton plant (see page 181). In sugar beet the first two (primary) leaves are physiologically quite different from subsequent (secondary) leaves, and differ markedly in susceptibility to fungal diseases (Russell, 1972) and aphids (Russell

and Barford, 1971). Primary and secondary leaves of many other crop species also differ in susceptibility to pests and diseases. It is therefore desirable to assess the resistance of breeding material at several different stages of growth, because tests at one stage may give results that do not apply to others.

Production of Resistant Varieties

Breeding for resistance to pests and diseases does not differ fundamentally from breeding for any other character (Allard, 1960); breeding methods that have been developed for other characters can therefore be used also for disease resistance. The most suitable methods to be used in a particular resistance breeding project will depend largely on the breeding system of the host plant

Table 2.1 EXAMPLES OF SELF- AND CROSS-POLLINATED CROP PLANTS

<i>Type of crop</i>	<i>Mainly self-pollinated</i>	<i>Mainly cross-pollinated</i>
Cereals	Wheat, barley, oats, rice, sorghum, millet	Rye, maize
Legumes ¹	Pea, soybean, peanut, sweet clover, subterranean clover	Alfalfa, red clover, white clover, alsike clover, runner beans (<i>Phaseolus</i> spp.)
Fruit ²	Peach, citrus	Blackberry, apple, banana, cherry, pear, plum, mango, date, raspberry, strawberry
Forage grasses	Mountain brome grass, soft brome grass, slender wheatgrass	Italian ryegrass, perennial ryegrass, meadow fescue, smooth brome grass, tall fescue, timothy
Starch, sugar, oil, fibre, etc ³	Cotton, flax, tobacco, pepper	Safflower, sunflower, sugar beet, sweet potato, castor bean, hemp
Vegetables	Lettuce, tomato, parsnip, Brassicae (broccoli, cauliflower)	Brassicae (Brussels sprouts, cabbage, kale, kohlrabi, radish, turnip) carrot, celery, cucumber, onion, parsley, spinach

¹ Both self- and cross-pollination occur in field bean (*Vicia faba*)

² Most fruit crops are generally propagated vegetatively

³ Both self- and cross-pollination occur in oil-seed rape (*Brassica napus*)

concerned, for example, whether it is cross-pollinated or self-pollinated, and on the sources of resistance that are available.

Table 2.1 lists some important crop species, classified according to whether they are mainly self- or cross-pollinated. The self-pollinated group of crop plants contains most of the important cereal crops of both temperate and tropical regions of the world, and also many important leguminous crops. On the other hand, most of the important forage grass species, vegetable crops and starch, sugar and oil crops are cross-pollinated. Several important crop species, including most types of fruit, potato and sugar cane, are normally vegetatively propagated, and the part of the plant harvested does not depend on the occurrence of successful pollination.

Although similar procedures are adopted in selecting for pest and disease resistance in self- and cross-pollinated crops, there are important differences in handling the two types of crop. In cross-pollinated crops, any individual plant which has been selected as being resistant cannot by itself usually form the basis of a variety, unless it can be propagated vegetatively on a large scale. There may be two primary reasons for this. First, many cross-pollinated plants are self-incompatible and cannot therefore be selfed. Second, if they are self-compatible, inbreeding usually causes a marked depression in vigour and yielding ability (**inbreeding depression**) in crops that are generally cross-pollinated. Normal levels of yield are recovered only when inbreds are crossed together in suitable combinations. This contrasts with the situation in self-pollinated crops, in which inbreeding does not usually significantly decrease either vigour or yield, and in which resistant varieties are each derived from an individual resistant plant.

Some of the main methods that have been used by plant breeders to produce resistant varieties of self- and cross-pollinated crops are briefly described below; for more detailed descriptions of these methods the reader is referred to general books on plant breeding, for example those by Poehlman (1959) or Allard (1960).

CROSS-POLLINATED CROPS

In cross-pollinated crops the most common method of breeding for resistance is **mass selection**, in which individual plants are selected for resistance from a heterogeneous population of plants; these selections are then allowed to inter-pollinate to produce seed for the next generation. The object is to achieve a higher proportion of resistant plants in each successive generation; by repeating the selection procedure in each generation (i.e. by **recurrent selection**), continuing improvements in resistance should be achieved.

In **line breeding**, selected plants are either selfed or interpollinated, and the resulting progenies or lines are individually tested for resistance; only the most resistant lines are retained for subsequent breeding. These are then interpollinated to produce a **composite cross**. An alternative method is to select resistant plants from a heterozygous population and to intercross these, or inbred lines derived from them, in all possible combinations in a **polycross**. The progenies of a polycross can be bulked, in which case resistant plants are subsequently selected from the bulk population, or the progenies of individual lines can be tested separately. Recurrent cycles of selection, for improved resistance and other desirable characteristics, can be carried out for as long as worth-while improvements continue to be obtained.

Resistant lines resulting from line-breeding or recurrent-selection programmes can be used to produce hybrid or synthetic varieties. A **synthetic variety** is produced by intercrossing a number of selected plants, lines or clones which have been found to have good combining ability. **Hybrid varieties** are produced by controlled pollination between lines; male sterility is often employed to achieve this (Poehlman, 1959). The parent lines of synthetic and hybrid varieties must be maintained separately so that the synthetic or hybrid varieties can be reconstituted as required. Only genotypes which produce superior hybrid combinations are put into synthetic varieties. This contrasts with simple mass selection procedures where progenies of selected plants are bulked without progeny testing or testing for combining ability, and with line breeding, where combining ability is not tested.

Plant breeders can produce resistant inbred lines of many cross-pollinated crop species by selfing or sib-mating (brother $\times$ sister mating) plants that have been selected for resistance. However, some species, including many Brassicas, have very effective self-compatibility systems and special techniques have to be employed for inbreeding. Some cross-pollinators such as hemp and cucurbits show little inbreeding depression, but the vigour of others such as alfalfa is so reduced, after two or three generations of inbreeding, that only very limited inbreeding is feasible.

SELF-POLLINATED CROPS

Three main breeding methods can be used in developing new varieties of self-pollinated crops: these are (1) **mass selection**; (2) **pure line selection**; (3) **hybridization**, which can be followed either by the pedigree or bulk selection methods or by a **backcrossing** programme. Each method leads to increasing homozygosity and the final result is a homogeneous, pure-breeding line, the plants of which are almost identical with one another in all heritable characteristics, including disease resistance.

Mass selection

In mass selection several plants of a similar phenotype are selected for resistance from a self-pollinating population and the progenies are bulked to form the basis of a variety. Individuals of such a variety may be similar morphologically but will differ in many other ways. For example, the component lines of a mass-selected wheat variety may have similar yields, heights and maturity dates but may differ in levels of resistance to certain diseases. Such varieties are easy to produce and their heterozygosity might give them certain advantages over pure line varieties, particularly in pest or disease resistance. In many developed countries, however, regulations for varietal purity and uniformity would preclude the release of cereal varieties developed by such a system of mass selection.

Pure line varieties

These are each derived from the progeny of a selfed homozygous plant selected from a line or commercial variety. The progeny is retested for resistance and evaluated for other desirable characters in succeeding generations and, if it is

considered sufficiently promising, is multiplied to produce a new variety. This is a very simple and quick procedure and many useful varieties of self-pollinated crops have been produced from 'off-types' which were selected and multiplied by particularly observant farmers or plant breeders. Such 'off-types' presumably arise either by mutations, occurring in a single plant, which are passed on to succeeding generations, or by natural hybridization with an unknown pollen parent. An early example of the production of a pure-line variety was the selection and multiplication of a single wheat plant that was taken from a field of Squareheads winter wheat by a farmer from Norfolk (England) more than one hundred years ago. The variety which resulted was called 'Browick' after the name of the village in which the first selection was made. Browick became widely grown in eastern England for many years and is a parent of several English winter wheat cultivars. It has expressed a high level of durable, adult-plant resistance to the yellow rust fungus, and attempts are being made to transfer this durable resistance to more modern, higher-yielding wheats.

Hybridization

This involves the artificial crossing of plants of two pure line varieties with the objective of combining desirable characteristics from each parent. This method enables the breeder to combine several forms of resistance to a particular parasite, or resistance to different pests or diseases, in a single variety. Plants in the F_1 generation from a particular cross are all genetically identical, but they are homozygous or heterozygous for individual resistance genes, depending on the genetic constitution of the parents. Genetic segregation occurs in the F_2 and later generations.

In all three breeding methods, homozygosity is regained in succeeding selfed generations. In the F_2 and later generations, selection for resistance to pests and diseases is usually based on one or two main methods, namely the **pedigree** or the **bulk population** selection methods. In the pedigree method, individual F_2 plants are selected for desirable features, including resistance to pests and diseases. The progenies of these selections are reselected in each succeeding generation until homozygosity is obtained (*Figure 2.1*). In the bulk population method, the early segregating generations (usually F_2 – F_6) are bulked together without selection. In later generations, when most plants are homozygous, individual plants are selected for resistance and their progenies are evaluated as in the pedigree method.

Although the bulk population method is easier and simpler to carry out than the pedigree method in the earlier generations, progenies of a very large number of plants must be grown for many generations before the numbers can be reduced by selection. For this reason the pedigree system is preferred by most plant breeders today, particularly if the heritable characters with which the breeder is concerned can easily be evaluated and selected for the early generations. Some aspects of both methods are combined in the **mass-pedigree** method, in which hybrid populations are grown in bulk until a pest or disease attack occurs naturally, when single plants are selected for resistance. These selections are then usually handled by the pedigree method.

In programmes of breeding for resistance to any parasite, the number of plants which should be evaluated for resistance and the number of plants which should

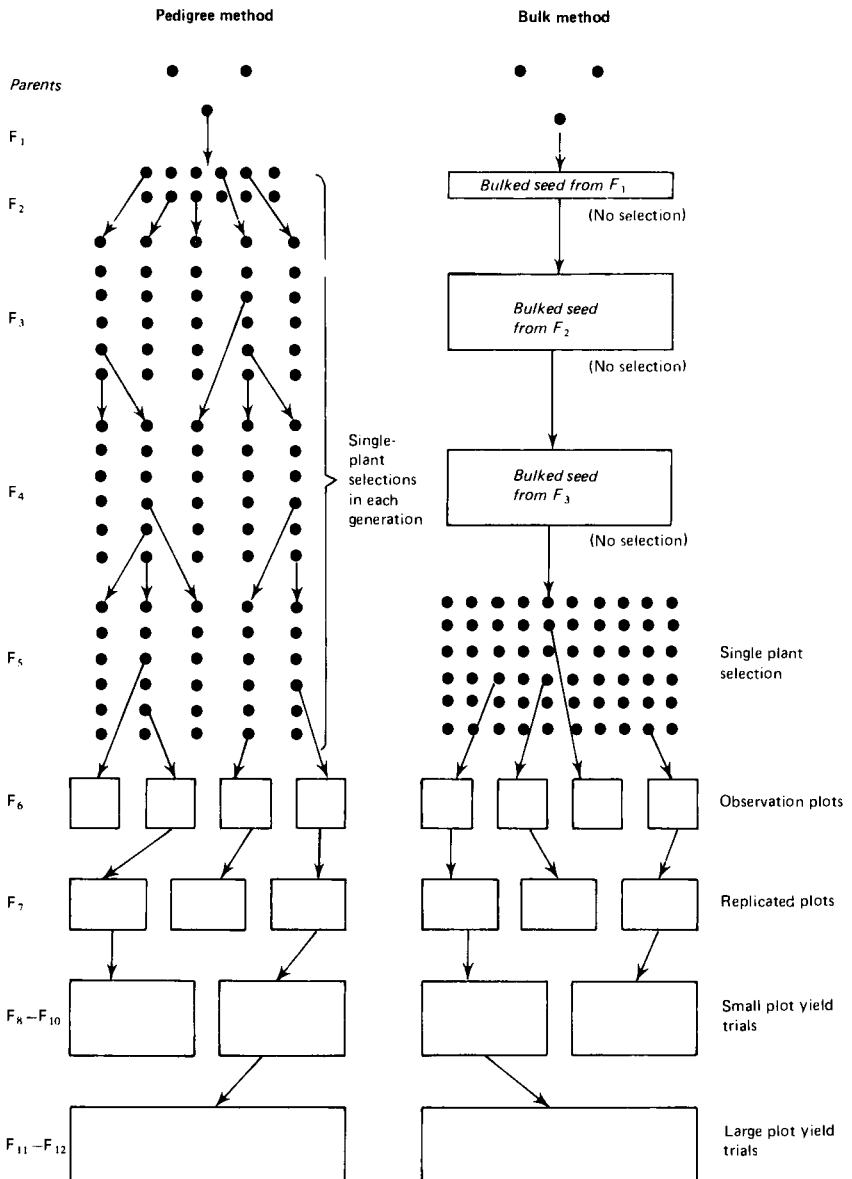


Figure 2.1 Flow diagrams of pedigree and bulk population methods of handling hybrid populations in a self-pollinated crop (After Briggs and Knowles, 1967)

be selected in each generation will depend largely on the complexity of the genetic system determining resistance. If the resistance is qualitatively inherited (for example, if it is controlled by a single, dominant gene) relatively few plants will need to be tested in each generation provided that the conditions for testing are satisfactory. With quantitatively inherited resistance, however, there may be

no clear-cut distinction between resistant and susceptible plants in the F_2 generation, and the expression of such resistance is often greatly influenced by environmental conditions. This can seriously hamper selection for resistance in the F_2 generation, in which there are usually very few plants available for testing, and selection often has to be deferred until the F_3 generation when more plants may be available. **Transgressive segregation**, where progenies of crosses are more resistant than either of the parental varieties, can occur with quantitatively inherited resistance; the chance of finding such progenies increases in proportion to the number of plants that are tested in the F_2 and later generations.

The main object of the **backcross method** is to transfer certain characteristics of a line or variety into breeding material. This method is particularly useful for transferring one gene or a few genes of large, easily identified effect, such as major genes conferring near-immunity to a parasite, from one genetic background to another. Specific resistance genes from wild species or agronomically unsuitable varieties have often been transferred in this way to susceptible but otherwise satisfactory varieties. Where resistance is a dominant character, plants of the two varieties are crossed and the progeny is backcrossed to the susceptible parent. The progeny of the first backcross generation is tested for resistance, and resistant plants are backcrossed to the susceptible (recurrent) parent. After several generations of such backcrossing, plants are obtained which are almost identical to the original susceptible parent except for the added resistance genes. The agricultural performance of such a variety should be unchanged unless the resistance genes concerned in the original resistant parent are closely linked with genes that condition undesirable characters. Where resistance is simply inherited but recessive, the progeny of each backcross generation must be selfed so that homozygous, recessive, resistant plants can be identified. The backcross method is not generally suitable for use with types of quantitatively inherited resistance which are controlled by polygenes.

VEGETATIVELY PROPAGATED CROPS

Several important crops are propagated vegetatively or asexually by means of tubers, cuttings, stolons, bulbs or other vegetative organs, or by grafting; these crops include sugar cane, potatoes, strawberries, raspberries, pineapples, bananas and most fruit trees and certain species of grasses. Plants of vegetatively propagated crop species are usually very heterozygous; vegetative propagation ensures that this heterozygosity is maintained by perpetuating the same genotype. This means that, once the breeder has achieved the desired level of resistance to a parasite in a vegetatively propagated crop, this level of resistance can be maintained without further selection while it is being multiplied. In this respect, vegetatively propagated crops resemble self-pollinated crops. Selection for resistance to pests and diseases is achieved, either by testing mixed populations of clones from different sources for resistance and selecting the best, or by selecting the best progenies obtained by hybridizing different clones. Because the parent clones are heterozygous, segregation occurs in the F_1 generation; each F_1 plant is potentially a new variety and must be tested separately for resistance.

Conclusions

The basic methods used in breeding for resistance are similar for crops with different breeding systems and for all types of parasites. Significant and rapid improvements in resistance can usually be achieved by quite simple and straightforward methods of testing and selection, although the detailed techniques that are most appropriate will depend on the crops and parasites concerned. Progress in breeding for resistance can be particularly rapid where improved resistance is the main breeding objective. However, resistance is usually only one of several breeding objectives; the breeder is forced to compromise between trying to achieve acceptable levels of yield, quality and other agronomically desirable characteristics, on the one hand, and trying to improve the level of resistance, on the other. He is therefore tempted to use those forms of resistance to pests and diseases which are the most simple to handle in a breeding programme. These forms are usually controlled by major genes and have often proved to be race-specific. The significance of this will be discussed in later chapters.

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The references cited in this chapter, together with those in Chapter 1, are listed in References—Part I, pages 42–44.

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PATHOGENIC FUNGI AND FUNGAL DISEASES

The Economic Importance of Fungal Diseases

Although there are more than 100000 different species of fungi, only about 200 of these are serious pathogens of crop plants. Most crop species are attacked by only a small number of fungi and very few of these cause severe and widespread economic damage. Nevertheless it has been estimated that fungal diseases are responsible for an annual reduction of more than 20 per cent of the total potential world food production from crop plants. The significance of such losses to mankind can be judged from the fact that about 70 per cent of man's food comes directly from crops and much of the remainder comes from animal food which is dependent on forage crops eaten by livestock; the latter crops can also be attacked by fungal diseases. Woodcock (1972) estimated that the monetary loss attributable to fungal diseases of crop plants exceeds US \$3 000000 annually.

Some fungal diseases can cause enormous losses of yield in particular crop species. Woodcock (1972) has estimated that more than 20 per cent of the potential yield of the potato crop is lost because of late blight. Fungal diseases of small-grain cereals also cause immense, world-wide losses. Wheat is damaged by many fungal diseases, the most important of which are the rust diseases which attack the leaves and stem, in spite of attempts to control them by growing resistant varieties and by the use of fungicides. Powdery mildew is the most severe disease of barley and can reduce grain yield by more than 50 per cent (see page 103). The yield of rice, which is the staple food crop of many tropical countries, can be drastically reduced by early attacks of the blast fungus *Piricularia oryzae* (see page 110).

Sugar beet can be very badly attacked, particularly in parts of the USA and southern Europe, by the leaf spot fungus *Cercospora beticola* (see page 126), and similar examples could be given of damaging fungal diseases in most crops; many of these will be discussed particularly in relation to breeding for resistance, in Chapter 4. Although it is impossible to quantify accurately the losses caused both directly and indirectly by fungal diseases of crop plants, it is clear that these losses are very great and every effort should be made to reduce them. Some of the indirect losses may not be apparent immediately but are nevertheless very significant. For example, many new varieties that are capable of giving increased yields have not been exploited fully because of their susceptibility to a disease. The risk of growing a very susceptible variety has sometimes been thought to be too great, possibly outweighing the potential benefit of higher

yields in the absence of disease. In such cases the breeders' efforts to develop more productive varieties have been nullified, not by a disease but by the threat of a disease. For example, in the UK in the early 1970s, a high-quality winter wheat variety, Maris Templar, which consistently gave higher grain yields than most of the contemporary varieties, was never grown over a large area because of its inherent susceptibility to certain races of the yellow rust fungus *Puccinia striiformis*. Farmers were thus deprived of a potentially more productive variety because of the indirect effects of a fungal disease.

Indirect loss from fungal diseases has also occurred because plant breeders have had to devote an excessive amount of effort to breeding for resistance to diseases. This has led to a corresponding reduction in effort directed towards improving important characteristics such as yield and quality. Much more work could be directed towards improving crop production if the major fungal diseases could be economically and effectively controlled. As the most efficient and cheap way of controlling diseases is through resistant varieties, more effective methods of breeding for resistance must be devised. The objectives must be to breed for types of resistance which are stable or **durable** (Johnson and Law, 1975), so that it is not necessary to develop new resistant varieties merely to replace those which have been attacked by resistance-breaking variants. The use of resistance which is highly race-specific has been very wasteful of resources and effort. The competition between the production, by the breeder, of new varieties with race-specific resistance, and the development of resistance-breaking races by a fungal pathogen, has almost always been won by the pathogen! It is, therefore, important to find ways of identifying and exploiting durable resistance to fungal diseases.

Other methods of overcoming problems of race-specificity should also be investigated and employed where they are advantageous. It has been suggested that multiline cereal varieties, each consisting of several genotypes with different major race-specific resistance genes, could be used to control rusts and powdery mildews. This concept of multiline varieties has recently been put to the test in the USA; the use of multiline oat varieties has significantly reduced damage caused by the crown-rust fungal disease (Frey, Browning and Simons, 1977). The use of multilines might extend the efficiency of certain types of race-specific resistance, and might be particularly useful in reducing damage from diseases where durable resistance has not yet been identified.

The role of the chemist in contributing to disease control should not be underrated. New, cheaper and more effective fungicides will undoubtedly play an important part in reducing damage by diseases so that the breeder can concentrate more on increasing the productivity of crop species. Nevertheless, because fungicides always give less disease control on very susceptible plants than on those which are even moderately resistant, it is important that new varieties are not unduly susceptible to any disease. However, breeders may have tried to achieve higher levels of resistance to fungal diseases than are necessary to avoid severe crop damage.

Some Characteristics of Fungi and Fungal Diseases

The basic life history of fungi is very simple. Spores germinate to give rise to one or more fine filaments, the **germ tubes**. These grow and give off branches each of which is known as a **hypha**. The collective term for a group of hyphae is a

mycelium which is the vegetative part of a fungus. The mycelium of the cereal powdery mildew fungus, *Erysiphe graminis*, grows on the surface of leaves of cereal plants (see Figure 4.6, page 109); absorption of nutrients from the host plant is effected by specialized feeding organs, the haustoria, which grow down into the epidermal cells. Some of the hyphae of the mycelium rise vertically from the leaf surface and spores (conidia) are budded off from them. The spores are released into the air to be carried to other parts of that plant or to other plants.

The mycelium of most other fungal pathogens is carried inside the organs of the host plant, although spore-bearing structures usually have access to the external environment to facilitate spore dispersal. In *Puccinia striiformis*, which causes yellow rust of wheat and barley, the hyphae grow subepidermally in the host, and uredospores are produced in uredosori which eventually rupture the epidermis so that they can be released into the air (see Figure 4.3, page 88).

A fungal spore germinates when it comes into contact with the surface of a host plant, provided that the environmental conditions are suitable. Important requirements for spore germination are high relative humidity of the surrounding air or a film of free water on the substrate, and appropriate light and temperature conditions. The physical nature of the surface with which spores are in contact is not important for germination; most fungal spores can be germinated on glass slides or on an agar gel. In some cases, however, spore germination is stimulated by compounds which are present on the surface of host plants. Russell and Evans (1968) found that substances which diffused from buds of young sugar beet plants into water stimulated germination of *Peronospora farinosa* conidia; this stimulation did not occur on other parts of the plant and these results are consistent with the greater susceptibility of buds of young plants to downy mildew. Conversely, germination of spores of other pathogens can be delayed or inhibited by compounds which are present on the surfaces of certain host plant genotypes. Examples of such inhibition of spore germination have recently been reported with *Puccinia striiformis* on wheat leaves (Russell, 1976a) and with *Erysiphe graminis* on barley leaves (Russell, Andrews and Bishop, 1976).

Entry of fungal pathogens into a host plant is either directly through the cuticle into an epidermal cell or through natural openings, particularly stomata, on the surface. Most rust fungi enter the host through the stomata and most powdery mildew fungi penetrate directly through the cuticle. Individual pathogens usually enter the host only through certain organs or parts of organs; most leaf-infecting pathogens will attack leaves but not flowers or roots, and most root parasites are confined to underground parts of the host. Infection by loose smut fungi, such as *Ustilago nuda* which attacks barley, occurs mainly through the floral parts; spores of the fungus are blown from infected plants to the stigma of a healthy plant where they germinate and a germ tube grows down inside the style to infect the ovary, much in the manner of pollen grains germinating before fertilization.

Plant pathogenic fungi fall into two main groups according to their food requirements. The first consists of the so-called **obligate parasites** (or **biotrophs**) which parasitize only living host plant tissues; the second group consists of **facultative parasites** (or **necrotrophs**) which kill the host cells before progressing through the tissues. Facultative pathogens can feed saprophytically and can therefore survive in the absence of living host plants. The work of Williams *et al.* (1967), Singleton and Young (1968) and Scott and Maclean (1969) has shown

that certain rust fungi can be cultured on artificial media; strictly speaking, therefore, these pathogens should be considered no longer as obligate parasites. Other pathogens, which today are classed as obligate parasites, will doubtless also be cultured artificially in the future. Nevertheless, outside the laboratory these fungi occur only on living plants; the distinction between biotrophs and necrotrophs therefore remains valid and is of crucial importance in breeding for resistance to fungal diseases. These two groups of pathogens differ in several important respects which are relevant to considerations of breeding for resistance.

OBLIGATE (BIOTROPHIC) PARASITIC FUNGI

These fungal pathogens usually obtain their food through specialized organs which are pushed into cells of susceptible host plants; it is through these **haustoria** that nutrients are absorbed from the host plant. The cytoplasm of the pathogen and host do not come into direct contact, but are separated by a complicated series of host and fungal membranes through which nutrients are transported. The complex nature of the haustoria of obligate fungal pathogens can be seen in electron micrographs of the haustoria of *Erysiphe graminis* such as those published by Bracker and Littlefield (1973).

Some obligate pathogens apparently obtain their nutrients without the aid of haustoria, by direct absorption through the intercellular hyphae, of nutrients from surrounding host cells. For example, in susceptible tomato varieties, the intercellular hyphae of *Fulvia* (*Cladosporium*) *fulva* are pressed closely against the cell walls of the host, thereby facilitating the uptake of nutrients (Lazarovits and Higgins, 1976). Intercellular hyphae of pathogens that do produce haustoria probably also can absorb limited amounts of nutrients from adjacent host cells.

The processes involved in the uptake of nutrients by obligate pathogens are not fully understood, but the osmotic pressure of the pathogen is presumably higher than that of the infected host cell if parasitism is to occur. A pathogen would therefore be expected to die, or at least not to flourish, where there is no adequate osmotic gradient between host and pathogen. There is some experimental evidence that high osmotic pressure of host cells is associated with effective resistance to obligate pathogens. Segments of wheat leaves containing a high concentration of sucrose, and which therefore presumably have a high osmotic pressure, are more resistant to yellow rust than are segments containing less sucrose. Similar results have been obtained with powdery mildew of barley (Russell, 1973) and with downy mildew of sugar beet (Russell and Evans, 1968). A direct relationship between the osmotic pressure of the cell sap of peach leaves and resistance to *Sphaerotheca pannosa* var. *persicae*, which causes powdery mildew, is suggested by the observations of Weinhold and English (1964). Selection for high concentrations of free sugars or a high osmotic pressure, particularly in epidermal cells, may therefore be worth while in breeding for resistance to some diseases.

Because most obligate fungal pathogens feed on living host cells through haustoria, it is to the advantage of these pathogens if infected cells are not quickly killed or severely damaged. It is not surprising, therefore, that in most of the rusts, smuts, powdery mildews and downy mildews, infected cells of

susceptible host plants are not visibly injured, at least until the pathogens have started to sporulate. In susceptible varieties of tomato, infection with *Fulvia fulva* does not cause even ultrastructural changes to the host cell until sporulation is extensive (Lazarovits and Higgins, 1976). In contrast, cells of resistant plants are often severely injured when they have been penetrated by haustoria; such injury is characteristic of the hypersensitive response to infection.

Obligate pathogens inevitably cause some damage to host cells even in a 'compatible' relationship between a host and a pathogen. This damage is caused mainly by the diversion to the pathogen of some of the nutrients and energy sources which would otherwise be available for the maintenance and growth of the host plant. Pathogens may also affect the pattern of growth of the host plant, either by producing growth substances themselves, or by affecting the concentrations of individual plant hormones in the host. Such effects are usually minor, however, in compatible relationships, and most damage to the host plant does not occur until the onset of sporulation. At this stage, the pathogen still further depletes the host plant of nutrients and often kills or damages cells around the points of sporulation. For example, in rust diseases the uredosori (sporing pustules) rupture the epidermis and cuticle so that uredospores can be released into the air. This not only destroys part of the epidermis but makes infected tissues liable to desiccation.

FACULTATIVE (NECROTROPHIC) PARASITIC FUNGI

Facultative pathogens kill cells of the host plant by secreting substances which either dissolve the cell walls or destroy the cytoplasm. *Corticium praticola*, which causes rotting of potato tubers, produces an enzyme which causes the cell walls to separate, with subsequent rapid death of the cells; these two effects apparently are part of one process (Hall and Wood, 1973). Enzymes of some other pathogens seem to cause separation of cell walls without injuring the contents of the separated cells. In such cases the death of host cells is thought to result from the secretion of some toxin by the pathogen.

The toxins of pathogens (**pathotoxins**) have been studied intensively, in particular those produced by species of *Helminthosporium*, a genus of the *Fungi Imperfecti* that includes the imperfect stages of many plant pathogens. *H. victoriae*, a very damaging pathogen of oats, produces victorin, a low-molecular-weight peptide linked with a nitrogen-containing sesquiterpene (Wood, 1967). Victorin causes extensive disruption of membranes in susceptible host cells, which die rapidly. The pathotoxin has little effect on resistant cells and its action is therefore very specific. Some races of *H. victoriae* do not produce victorin and are not pathogenic. Pathogenicity therefore depends on the genotypes both of fungus and of host plant. Similar specific interactions between host and pathotoxins occur between maize and *H. carbonum*, which causes leaf spot, and between maize and *Cochliobolus* (*Helminthosporium*) *heterostrophus*, the southern corn leaf blight fungus.

It is important to differentiate between the two types of disease susceptibility involved; the first is susceptibility to attack by the pathogen itself, and the second is susceptibility to damage caused by the toxin which that pathogen produces. Thus, maize plants which do not have the Texas type of cytoplasm (see page 113) can become infected with race T of *C. heterostrophus*, but

they are not severely damaged because their cells are not sensitive to the toxin that is produced by this race of fungus. On the other hand, this toxin very severely damages cells of maize plants which do have the Texas cytoplasm. Infection with race O of *C. heterostrophus*, which also produces a pathotoxin but only in very low concentrations, does not cause severe damage to plants with either 'normal' or Texas cytoplasm. Susceptibility to the pathogen is therefore quite distinct from sensitivity to the pathotoxin. In breeding for resistance to a disease that is caused by a pathotoxin-producing fungus, the breeder can choose whether to breed for resistance to the pathogen or for tolerance of the pathotoxin. The two kinds of resistance involve quite different resistance mechanisms and are independently inherited. Resistance to *C. heterostrophus* in maize is under the control of nuclear genes, whereas tolerance of the pathotoxin is inherited cytoplasmically. As tolerance to pathotoxins seems often to be very race-specific, the breeder might be wise to select, not only for tolerance to the toxin, but also for resistance to the pathogen.

Variability in Fungal Pathogens

Individual genotypes of a fungal pathogen can differ from each other in many inherited characteristics, for example in morphology, physiology and pathogenicity (Person and Ebba, 1975). In this respect fungi are no different from other groups of organisms. Most of these characteristics are governed by nuclear genes arranged on chromosomes which behave in much the same way at mitosis and meiosis as do those of other organisms (Fincham and Day, 1971). Some features, however, are controlled cytoplasmically (Jinks, 1966).

The variability which is of particular importance to the plant breeder is that which is concerned with the development and dissemination of new physiologic races, with the ability to attack varieties that previously were resistant. These physiologic races are often called 'resistance-breaking' races although, as Day (1974) points out, the resistance of the varieties does not break down, in the strictest sense, because it is still as effective as it ever was against the original forms of the pathogen; however, it is ineffective against the new form of the pathogen.

It is important to emphasize that a particular physiologic race of a pathogen may comprise many different genotypes. These genotypes have only one essential factor in common, which is the possession of a virulence gene able to overcome resistance controlled by a corresponding gene in the host. Thus a single physiologic race may consist of individuals which have a virulence gene in common but differ from one another in morphology, physiology and virulence towards other host plants with other resistance genes. This section describes the means whereby resistance-breaking races of pathogens can arise, and their relevance in breeding for disease resistance.

New races can arise in four main ways: (1) by mutation in somatic cells; (2) by recombination of nuclear genes during sexual reproduction; (3) by reassortment or exchange of genetic material in somatic cells, and (4) by mutation of extrachromosomal or cytoplasmic genes.

SOMATIC MUTATIONS

The rate at which new variants of a pathogen are produced will depend on the mutation rate of genes at a particular locus. Although this rate varies greatly

from gene to gene and from pathogen to pathogen (Robinson, 1971), Day (1974) suggests that the natural mutation rates of genes in most pathogens are sufficient to generate mutants which can attack any host variety protected by only a single resistance gene.

A mutant of a pathogen with **virulence** towards a resistance gene of a previously resistant host variety will quickly become established in the pathogen population if that variety is widely grown (Van Der Plank, 1963). In the absence of that variety, however, the fate of a new physiologic race will be determined partly by its ability to compete successfully with other genotypes of the pathogen, i.e. its fitness to survive. Genes for virulence are sometimes associated with reduced fitness (Van Der Plank, 1968), which is to be expected because most mutations are harmful or even lethal. However, some mutations for virulence are obviously not very harmful because they continue to survive in pathogen populations in the absence of selection for virulence on an appropriate host genotype. Even where a mutation for virulence is associated with one or more disadvantages, selection pressure can be so strong in favour of races with new virulence on hosts with a corresponding resistance gene, that any lack of fitness would soon be overcome; there would be no competition from other pathogen variants and the virulent genotype would therefore be able to flourish and multiply, allowing selection for increased fitness. Such selection seems to have occurred in resistance-breaking races of *Erysiphe graminis*. The gradual accumulation of virulence genes or alleles in individual isolates of *E. graminis* f.sp. *hordei*, giving rise to complex races, does not appear to affect the vigour of the pathogen (Wolfe, 1972).

SEXUAL REPRODUCTION

Races of fungal pathogens which carry new genes for virulence can be perpetuated and disseminated asexually. Resistance-breaking races of *Puccinia striiformis* have developed and spread quickly and extensively although this fungus has no known sexual stage. Sexual reproduction is not an important part of the life cycle of *Phytophthora infestans* in most parts of the world, but the fungus has been notorious for its ability to develop new resistance-breaking races. It is clear, therefore, that sexual reproduction in fungal pathogens is not necessary for the formation of new physiologic races. Nevertheless, sexual reproduction does promote recombination of genes at meiosis by means of random assortment of chromosomes and the crossing-over of parts of chromosomes. Although this does not create new genes, it allows the development of variants with new combinations of virulence genes and of genes for increased fitness.

SOMATIC RECOMBINATION OF GENES

Many important fungal pathogens have heterokaryotic cells which carry two or more genetically different nuclei. For example, the Basidiomycetes, which include the rust and smut fungi, have binucleate cells. In certain fungi the heterokaryotic condition is only temporary and often follows anastomoses between cells of adjacent hyphae, which soon return to the normal mononucleate condition. In some fungi, cells are almost always mononucleate.

When conidia (asexual spores) develop from a heterokaryotic hyphal cell, the segregation of dissimilar nuclei in the spores encourages genetic variation (Day, 1974). Anastomoses between hyphae of different pathogen genotypes can also generate variability. In *Puccinia striiformis*, anastomoses between hyphae of two different physiologic races have been shown to give rise to a third race (Little and Manners, 1969; Goddard, 1976).

Diploid nuclei are sometimes formed in heterokaryotic cells by the fusion of haploid nuclei which carry different genetic material. Reassortment and recombination of genes can occur in such nuclei at mitosis and this has been called the **parasexual cycle**. Parasexuality is probably important in generating and stabilizing new genetic variation in pathogenic fungi, particularly in those without sexual reproduction (Tinline and MacNeil, 1969; Day, 1974). It has been suggested that the parasexual cycle may be the main source of variation in a number of important plant pathogens, including *Piricularia oryzae* (rice blast), *Helminthosporium sativum* (foot rot of barley) and *Cochliobolus heterostrophus* (southern leaf blight of maize); the existence of resistance-breaking races of these pathogens has caused serious difficulties in breeding for resistance to these diseases.

EXTRACHROMOSOMAL VARIATION

Several examples of cytoplasmic inheritance of important characteristics, such as growth rate and virulence, have been demonstrated in fungi (Jinks, 1966). Virulence of *P. graminis* f.sp. *avenae* (stem rust of oats) to oat varieties carrying resistance gene *E* is maternally inherited (Green and McKenzie, 1967) and may be controlled by a single plasmagene (Johnson, Green and Samborski, 1967). The possibility of cytoplasmic inheritance of virulence does not seem to have been examined in most fungal pathogens and more research on this subject is strongly indicated.

Virulence in fungal pathogens is usually a recessive character. With the removal of selection pressure in favour of virulence, when host plant varieties with corresponding resistance genes are withdrawn, dominant genes for avirulence would probably become prevalent in the pathogen populations concerned; in other words, there would be a reversion to the former state of avirulence (Person and Ebba, 1975). However, the recessive genes for virulence would still be present in the pathogen populations and could become common again if selection pressure in favour of virulence was reapplied.

The way in which a pathogen is dispersed is important in determining the rate at which any resistance-breaking races will be disseminated. New races of pathogens with air-borne spores will spread more quickly and over a wider area than those of soil-borne fungi. It might be expected, therefore, that there would be a more rapid and widespread breakdown of resistance to pathogens such as *Phytophthora infestans* or *Erysiphe graminis* which produce enormous numbers of air-borne conidia, than to those such as *Synchytrium endobioticum* (the potato wart disease fungus) which are soil-borne. In practice, resistance-breaking races of most soil-borne fungi have not been a serious problem whereas the rapid dispersal of resistance-breaking races of rusts and mildews with air-borne spores has often led to the abrupt withdrawal of many otherwise excellent cereal varieties.

Types of Resistance

The main aim of breeding for resistance is to produce varieties which have less disease or which suffer less damage from disease. In practice, resistance to a fungal disease may comprise several kinds of resistance; these include resistance to the pathogen itself, disease escape or tolerance of the effects of disease as shown in *Figure 3.1*. Although the term 'resistance' is commonly used to

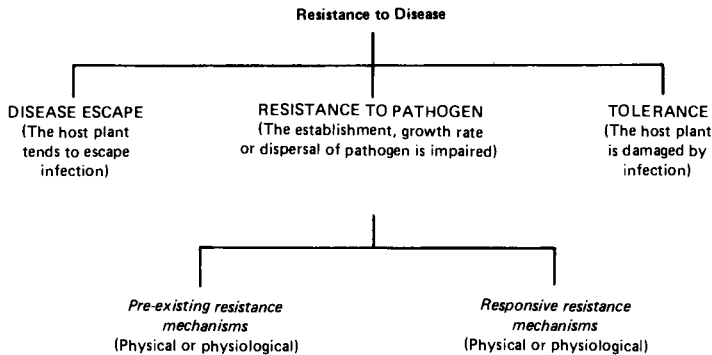


Figure 3.1 A classification of different theoretical types of host-plant resistance to fungal diseases

refer only to direct resistance of the host plant to the pathogen, a broader concept of resistance would seem to be more appropriate and useful to the practical plant breeder. **Resistance** is therefore used in this book in a very broad sense and embraces many different inherited factors.

DISEASE ESCAPE

Disease escape can take many forms, some of which may give a very effective control of disease in the field. For example ergot, which is a fungal disease of inflorescences of cereals caused by *Claviceps purpurea*, does not affect those varieties of wheat and barley in which the flowers remain closed until pollination has occurred. In such varieties the spores have no opportunity to enter the flowers and infect the stigma when it is at a susceptible stage. However, ergot infection can easily be achieved in these varieties by introducing spores of *C. purpurea* into the still-closed flowers at or about the stage of anthesis. The closed-flowering habit is a very good example of resistance involving a disease escape mechanism where a barrier to inoculation prevents close contact between a pathogen and its host. If such a barrier becomes ineffective for any reason, the host plant in question is susceptible to attack.

Another form of disease escape which is based on morphology can occur with *Septoria nodorum* on wheat. Spores of *Septoria* are usually splash-dispersed from leaf to leaf in a crop by rain drops, thus infecting successive leaves on the stem. However, in wheat varieties which have tall straw with long internodes, splash dispersal of spores is less effective than in shorter-strawed varieties (Sharp, Brönnimann and McNeal, 1972; Scott, 1973). The upper leaves of tall-strawed

varieties therefore remain relatively free of *Septoria* under conditions of natural spread of disease in the field although they are often very susceptible to *Septoria* when artificially inoculated.

An erect growth habit can contribute to disease escape in the powdery mildew disease of cereals. Fewer spores of *Erysiphe graminis*, the causal fungus, were found to be deposited from a spore cloud on barley plants with erect leaves than on those with prostrate leaves, decreasing the number of infections (Russell, 1975a). An erect growth habit in wheat similarly can contribute to escape from the yellow (stripe) rust disease caused by *Puccinia striiformis* (Russell, 1975b).

Another kind of disease escape is shown by cereal plants on whose leaves spores of fungal pathogens do not germinate readily. The germination of uredospores of *Puccinia striiformis* is inhibited on the leaves of some winter wheat varieties, notably Holdfast (Russell, 1976a) and a smaller proportion of germinated uredospores penetrate stomata and form substomatal vesicles in Holdfast than in several other varieties (Russell, 1976b). Thus two kinds of disease escape seem to be shown by leaves of Holdfast.

RESISTANCE TO A FUNGAL PATHOGEN

Resistance to a fungal pathogen concerns any inherited characteristic of a host plant which impedes the establishment or growth of that pathogen. This is quite distinct from disease escape where inherited factors discourage cell-to-cell contact between host and pathogen. Thus a cuticle or epidermal cell wall that cannot be penetrated by a pathogen might reasonably be considered to be a disease escape mechanism. Conversely an impenetrable wall of an internal cell could be classified as resistance to the pathogen because the host plant itself would have been invaded.

The establishment of a fungal pathogen can be inhibited by resistance mechanisms which already existed before inoculation ('pre-existing' mechanisms) or by those which are activated by inoculation ('active' or 'responsive' mechanisms). Pre-existing barriers to establishment may be physical or physiological. Certain pathogens are unable to colonize host cells with heavily lignified walls, presumably because these act as a physical barrier. However, they may constitute a physiological barrier also, because the walls may not be easily degradable by the pathogen's enzymes; in addition, lignified walls may contain fungitoxic compounds such as ferulic acid (Hartley, Jones and Wood, 1976; Hartley, Harris and Russell, 1978). Differences in osmotic pressure between host and pathogen may also be a physiological barrier to the establishment of a pathogen in the tissues of a host plant. For example, the fungus which causes powdery mildew in peach can apparently only become established and grow normally if the osmotic pressure of the host cell is significantly below that of the mycelium (Weinhold and English, 1964). Such physiological barriers may be common because factors which increase the osmotic pressure of host plant cells apparently inhibit the development of many fungal pathogens including *Erysiphe graminis* in barley, *Puccinia striiformis* in wheat (Russell and Hudson, 1973) and *Peronospora farinosa* and *Erysiphe betae* in sugar beet (Russell and Barford, 1971).

There is very little published information concerning pre-existing resistance mechanisms. This may suggest that they are not important, but it seems more likely that insufficient work has been carried out to demonstrate their potential importance.

Most of the published work concerning resistance to the establishment of fungal pathogens relates to responsive mechanisms that prevent attempts by the pathogen to penetrate host cells. One mechanism is the rapid deposition of compounds on the cell wall at or near the point of attempted penetration. Compounds deposited in this way include callose (Hardwick, Greenwood and Wood, 1971; Heath, 1971; Bracker and Littlefield, 1973), lignin (Hijwegen, 1963; Friend, 1973; Ride, 1975; Friend, 1976) and an unidentified basic-staining material (Lin and Edwards, 1974). The role of these and other compounds in restricting hyphal penetration is uncertain, but many of them may act both as a physical and as a chemical barrier to the pathogen.

Hypersensitivity

Host plant cells that have been penetrated by a pathogen often die rapidly as a result of **hypersensitivity**. Pathogens that elicit a hypersensitive response in cells of a particular host genotype are said to be **incompatible** with that host. This hypersensitive response is very complex and has been the subject of intensive study. Hypersensitive cells produce compounds, including phytoalexins, many of which are phenolic; these are not only fungitoxic but also strongly autotoxic so that, in effect, the cells kill themselves (Cruickshank, Biggs and Perrin, 1971; Deverall, 1976). A pathogen may die as a result of two distinct mechanisms; either directly by the action of the host plant toxins, or indirectly because the host cells die and are unable to support an obligate pathogen. Much recent work has suggested that hypersensitivity is the result, rather than the cause, of resistance (Ogle and Brown, 1971; Kiraly, Barna and Ensek, 1972; Mayama *et al.*, 1975). On the other hand, many workers consider that hypersensitivity can directly cause incompatibility (Bracker and Littlefield, 1973; Maclean *et al.*, 1974).

Yamamoto and Matsuo (1976) suggest that interactions between the DNA of specific genotypes of host and pathogen may be a mechanism whereby pathogens can recognize host plants. Such interactions may result in a hypersensitive reaction which would be a cause rather than a consequence of resistance. Although the precise nature of hypersensitivity is not understood, this kind of resistance has been widely used by plant breeders, mainly because it is easy to exploit in a breeding programme. It is usually controlled by a few major dominant genes, and resistant plants can easily be distinguished from susceptible plants. Differences in resistance involving hypersensitivity tend to be clear-cut and qualitative; this greatly facilitates breeding for resistance.

Resistance based on hypersensitivity has given a very effective, although often temporary, control of many fungal diseases, for example late blight of potatoes and powdery mildew of barley. The temporary nature of the resistance is due to the development and dissemination of resistance-breaking races of the pathogens concerned. Varieties of potatoes with *R* genes for hypersensitivity to the blight fungus, *Phytophthora infestans*, are susceptible to isolates of the pathogen that do not trigger off a hypersensitive response in such varieties

(see page 118). Similarly, barley varieties with hypersensitive resistance to some isolates of *Erysiphe graminis* have been severely attacked by other isolates (see page 104). There is obviously a very strong selection pressure in favour of these resistance-breaking races and they become predominant in the pathogen population.

Hypersensitivity seems to involve a 'lock and key' situation in which there is a specific interaction between particular genes of host and pathogen. This has given rise to the concept of a gene-for-gene relationship between host and pathogen (Flor, 1956). This hypothesis, which has been developed and extended by many other workers, has been reviewed by Sidhu (1975). Such gene-for-gene relationships have been implicated in most cases of 'resistance breakdown', and the avoidance of those types of resistance which involve specific interactions between host and pathogens may therefore increase the durability of resistance.

Other types of resistance to the pathogen

Although hypersensitivity is the most common form of resistance affecting the establishment of a fungal pathogen in the host, other resistance mechanisms may also inhibit the establishment of a pathogen. Penetrated cells may be killed in other ways than by the actions of toxins. For example, pectic enzymes from the pathogen may injure the host cells because enzymically degraded cell walls cannot support the limiting membrane (plasmalemma) of living cells, so that the membrane is damaged (Basham and Bateman, 1975). Cells of adult plants of certain winter wheat varieties die prematurely when penetrated by *Puccinia striiformis* but neighbouring cells appear to be unaffected. In such cases there are usually no visible signs of chlorosis or necrosis on inoculated leaves, and other mechanisms than hypersensitivity seem to be implicated.

Many inherited factors can affect the development of fungal pathogens in the tissues of the host plant after the pathogens are established. A series of attempts by a fungus to colonize host plant cells, which are partly unsuccessful because of hypersensitivity or other resistance mechanisms, will greatly decrease the growth rate of the pathogen and may eventually kill it. Heavy lignification of cell walls in the tissues, which may be present before infection or produced in response to infection, can restrict or delay the growth and spread of a pathogen. Lignin-like polymers, which occur in healthy mature wheat plants, are similar to compounds which are formed in response to inoculation with non-pathogenic fungi (Ride, 1975). Adult plants are usually more resistant than younger plants to foliar pathogens; this adult-plant resistance may be caused partly by an accumulation of lignin-like compounds in leaf tissues. Hartley, Harris and Russell (1978) have shown that there are higher concentrations of such compounds in parts of healthy wheat flag leaves which are resistant to *Puccinia striiformis* than there are in more susceptible parts of the leaves.

The growth rate of a pathogen may be decreased by the presence of antibiotic compounds in the tissues of the host: these may also be pre-existing or formed in response to infection. Certain free amino acids, either alone or in combination, are known to be toxic to micro-organisms. For example, high concentrations of certain free amino acids are associated with non-hypersensitive resistance to some fungal pathogens of wheat and barley although no causal

relationship has been found (Russell and Hudson, 1973). Conversely, low concentrations of other free amino acids are associated with resistance to these pathogens, perhaps because of a shortage of amino acids which are essential for their rapid growth and, in particular, for protein synthesis.

With facultative pathogens, which do not usually feed on living tissues and are therefore necrotrophic, pathotoxins are released from the leading edge of the mycelium and these kill the host tissues in advance of the pathogen. Any features of a host plant which inactivate either these pathotoxins or the enzymes that subsequently degrade the killed tissues, will restrict the growth of such pathogens. The mechanisms of resistance to many important diseases, including southern corn leaf blight of maize and Victoria blight of oats, which are caused by necrotrophic fungi, have been studied in detail. *Cochliobolus heterostrophus* produces a toxin in maize varieties which carry the T cytoplasmic factor for male sterility, and this toxin affects the membranes of the mitochondria of host cells causing them to swell irreversibly (Miller and Loeppé, 1971), whereas maize varieties with other types of cytoplasm are unaffected. *Helminthosporium victoriae*, which causes Victoria blight of oats, also produces a toxin but this acts on the plasma membrane of cells of susceptible, but not of resistant, host plants (Samadder and Scheffer, 1968).

Resistance to both facultative and obligate pathogens is often expressed as an increased generation time of the pathogen, i.e. the period between the time of inoculation and the onset of sporulation is prolonged. Russell and Muszanskyj (1976) have shown that the generation time of *Erysiphe graminis* is longer on resistant than on susceptible barley varieties, and on adult than on young barley plants of the same genotype. Even small increases in generation time of a fungal pathogen can significantly delay the development of a disease epidemic in the field. A prolonged generation time can therefore be a very important criterion in selecting for resistance to a fungal disease. An increased generation time can be attributable to any factor, or combination of factors, which delays or alters the normal growth pattern of the fungus. These factors include hypersensitivity, delayed or reduced spore germination and interference with the establishment of the pathogen, in addition to mechanisms which directly inhibit fungal growth. In spite of the many different causes of increased generation time, it is easy to compare the generation time of a particular fungus on different host genotypes by recording the time interval between inoculation and the start of sporulation in artificial inoculation tests.

The number of spores of a fungal pathogen produced per unit area or volume of infected host plant tissue is usually partly under the genetic control of the host although it can be greatly influenced by environmental conditions. Fewer spores are usually produced on resistant than on susceptible plants, for a variety of reasons. Any inherited factors of the host plant which inhibit the growth of the pathogen will result in reduced sporulation. A tendency to escape infection will reduce the number of infections, and the number of spores which are subsequently produced on an infected plant will usually be decreased. Reduced sporulation could be attributable either to a smaller area of sporulating tissue or to fewer spores per unit area of diseased tissue. In rust and powdery mildew diseases, where sporulation usually occurs in discrete pustules, the number of pustules per unit area of leaf can be counted or assessed using a simple key (see page 76). Alternatively, pustules can be examined under the microscope in order to estimate the mean number of spores per pustule. This method has

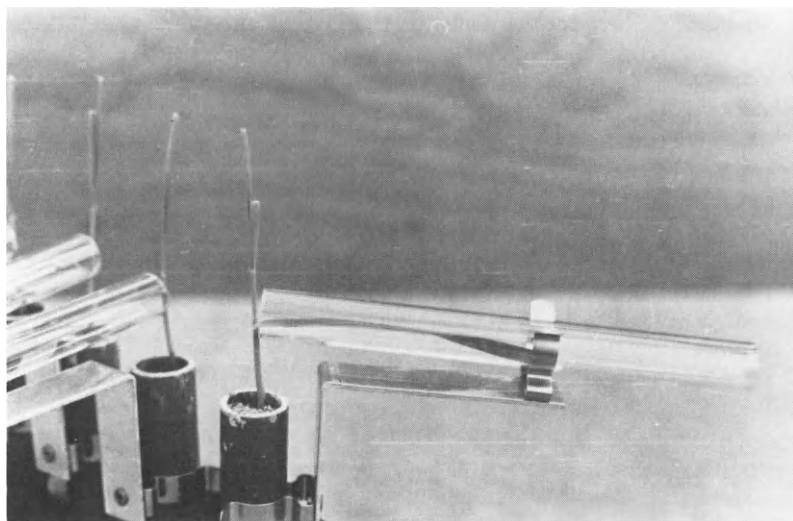


Figure 3.2 The spore production by *Puccinia striiformis* on individual wheat leaves can be measured accurately by collecting them in a glass tube and weighing them on a microbalance. (By courtesy of Dr R. Johnson, Plant Breeding Institute, Cambridge)

been used in comparing sporulation of *Erysiphe graminis* on barley (Russell, Andrews and Bishop, 1976) and *Puccinia striiformis* on wheat (Russell, 1976b). Even more accurate comparisons of spore production of *Puccinia striiformis* on different wheat genotypes can be made by weighing the spores produced on individual leaves (Figure 3.2) on a microbalance (Johnson and Bowyer, 1975; Johnson and Taylor, 1976).

TOLERANCE

Plants that are diseased to the same extent as other plants but are not damaged as much by the infection are **tolerant** to that disease. Tolerant plants do not affect the development of the pathogen concerned, which in turn has little effect on them. Although tolerance has often been used by plant breeders in the case of virus diseases, tolerance to fungal diseases has been much neglected. There is no doubt, however, that tolerance to fungal pathogens is a widespread phenomenon, having been demonstrated experimentally in a number of cases including wheat stem rust (Calpouzos *et al.*, 1976) and barley with powdery mildew (Little and Doodson, 1972). Many plant breeders and plant pathologists are critical of the use of tolerant varieties because such varieties may provide a reservoir of infection for other less-tolerant varieties. Although tolerant varieties are no more likely to become infected than others, profuse sporulation of pathogen can occur on them so that they may become serious sources of infection for other varieties, particularly if they are grown on a large scale. It is probably desirable, therefore, that tolerance to fungi should be used only in conjunction with other forms of resistance, to avoid this possibility. Tolerance would then act as an additional safeguard against heavy losses from a disease; this would be particularly valuable where a 'breakdown' of other forms of resistance is likely.

Tolerance to a fungal disease is less easy to assess than most other kinds of resistance. Tolerance can be manifested in many ways, for example by a smaller effect of disease on the growth rate of shoot or root growth or on the yield of seed. However, the breeder is mainly concerned with the effect of tolerance on the agriculturally important end-products of the crop. The breeder of small-grain crops will therefore be interested mostly in the effects of disease on the yield of grain when selecting for tolerance. If, however, he is breeding for tolerance to a disease of a forage crop, he will be concerned primarily with the effects of the disease on the yield of fodder; yield of seed will be of secondary importance. In selecting for tolerance in early generations of a breeding programme, plants which yield well in spite of being diseased should be selected and bred from. In later generations, when tests on a larger scale are possible, yield trials with infected and uninfected plots should be laid down to test the effects of the disease on yield. Such trials can consist of adjacent inoculated and non-inoculated plots in areas where the disease in question is unlikely to spread from plot to plot. It is often necessary, however, to control the disease on the non-inoculated plots by repeated applications of appropriate fungicides. If a disease is endemic in the area where a test for tolerance is to be carried out, it is not usually necessary to inoculate any plots but the disease must be controlled by fungicides on some of the plots.

Sources of Resistance

As a general rule, the best sources of resistance are likely to be those in locally adapted, high-yielding varieties because they can easily and quickly be exploited in a breeding programme. Slightly less useful sources are exotic varieties from other parts of the world, and indigenous varieties which have become outclassed and superseded by new varieties. It is important to avoid any source of resistance which, because of a race-specific relationship between host and pathogen, has been involved in a previous 'breakdown' of resistance to the disease concerned. Sources of resistance from other species are difficult to use, even when fertile progeny from interspecific crosses can be obtained. It is usually impossible to transfer polygenically controlled disease resistance by interspecific crosses, and simply inherited types of resistance from other species have usually been race-specific and therefore not stable or durable. Nevertheless, resistance to a few diseases has been successfully transferred from related species to breeding material from which resistant cultivated varieties have been derived (Watson, 1971), for example resistance to *Fusarium* wilt in tomato (see page 131).

Sources of resistance to a disease can sometimes be 'induced', or, more accurately, made to occur more frequently, by mutagenic chemical or radiation treatments of the host plant. Jørgensen (1971) reported that several barley lines carrying mutations which had been induced independently in several different barley varieties, were resistant to powdery mildew, caused by *Erysiphe graminis* f.sp. *hordei*. This resistance is conditioned by a single recessive gene, *ml-o*, which had not hitherto been identified. This gene has subsequently been found in untreated barley populations (Jørgensen, 1973), suggesting that so-called 'induced' mutations for disease resistance usually occur naturally, albeit at low frequencies. Nevertheless, induced mutations can be an effective way of increasing the frequency of genes for resistance which occur only rarely in

natural populations (Sigurbjörnsson and Micke, 1974; Grundewaldt and Grundewaldt, 1977).

If no suitable source of resistance to a disease has already been found, local and exotic varieties and related species will have to be screened for resistance. These screening tests must be carried out under conditions that will allow differentiation between different levels and kinds of resistance in the material. These conditions will vary according to the crop species and the types of pathogen which are involved.

The Inheritance of Resistance

The mode of inheritance of resistance to a disease will largely determine which methods of breeding and selection to use. It is therefore of great importance for a plant breeder to discover at the start of a breeding programme whether the resistance is controlled by a few or many genes, and whether the influence of the cytoplasm is likely to be significant. It is also important to know whether resistance is dominant or recessive to susceptibility.

MONOGENIC RESISTANCE

There are many advantages in handling monogenic resistance, which is controlled by a single gene, particularly if it is dominant. Differences between resistant and susceptible plants are clear-cut, and segregation for resistance occurs in simple ratios. Unfortunately, it has been the general experience that monogenically controlled resistance to diseases is race-specific and has usually soon ceased to control them effectively because of the occurrence of resistance-breaking races of pathogens.

OLIGOGENIC RESISTANCE

Oligogenic resistance is controlled by a small number of major genes each of which has a large effect. Such resistance can involve either a complex of different resistance mechanisms, each of which is conditioned by a single gene, or a single mechanism which is controlled by several genes. These two types of oligogenic resistance have not often been distinguished although there are potentially important reasons for doing so. A combination of different resistances, even if they are each controlled by a single gene, may be more difficult for a pathogen to adapt to than a single resistance mechanism.

POLYGENIC RESISTANCE

The term **polygenic** is used for types of resistance which are known to be controlled by many genes. Such resistance can involve a single but complex mechanism, such as a disease-escape factor or heavy lignification of the cell walls, which is the end-product of several distinct chemical reactions. Alternatively, polygenic resistance can be caused by a number of independent

mechanisms, some of which may be controlled by major genes and others by minor genes. It is clear, therefore, that there are quite different forms of oligogenic and polygenic resistances and that these will have different implications in breeding for resistance. Unfortunately, very few examples of polygenically controlled resistance have so far been analyzed in sufficient detail for the individual resistance mechanisms to be characterized. Nevertheless, resistance that is conditioned by many genes has usually been more durable and less race-specific than resistance that is controlled by only a few genes. This conclusion is supported by the results of breeding for resistance to fungal diseases of several crops, described in Chapter 4. This advantage of stability or durability of polygenic resistance is offset by the disadvantage of the complex nature of the resistance, which makes it difficult to exploit in a breeding programme. Differences in this type of resistance tend to be quantitative rather than qualitative but, provided that suitable screening and selection methods are used, very satisfactory levels of resistance can be achieved. These methods will be discussed later in this chapter.

CYTOPLASMIC INHERITANCE OF RESISTANCE

There are few published references to the influence of cytoplasmic genes on resistance to fungal diseases. Probably the best documented example of the cytoplasmic inheritance of susceptibility to a disease is the reaction of maize to *Cochliobolus heterostrophus*, the fungus which causes southern corn leaf blight. As described on page 113, certain variants of this pathogen can attack maize varieties with the Texas type of cytoplasm, but cannot attack those with other types of cytoplasm. These variants caused severe damage to maize varieties with T cytoplasm in the USA in 1972 and 1973 (see page 116). Experiments involving reciprocal crosses between resistant and susceptible host genotypes have sometimes suggested a maternal or cytoplasmic influence on the expression of resistance in other host plant-pathogen combinations, for example with wheat and *Puccinia striiformis*. Unless such reciprocal crosses are made and compared for degree of resistance to disease, the influence of the cytoplasm will not be detected. Reciprocal crosses are not always made, because of the extra work involved, and the importance of the cytoplasm on the expression of disease resistance may therefore have been seriously underestimated.

Testing and Selecting for Resistance

Wherever possible, screening and selection tests for resistance to fungal diseases should be carried out in the field. There are two main advantages of field tests over glasshouse and laboratory tests; much larger populations can be dealt with in the field, and plants are tested for resistance under natural conditions. Nevertheless, laboratory and glasshouse tests can play an important part at some stages of most breeding programmes, particularly because conditions can more easily be standardized than they can be in the field. For example, testing for resistance to *Erysiphe graminis* can be carried out using segments of detached barley leaves

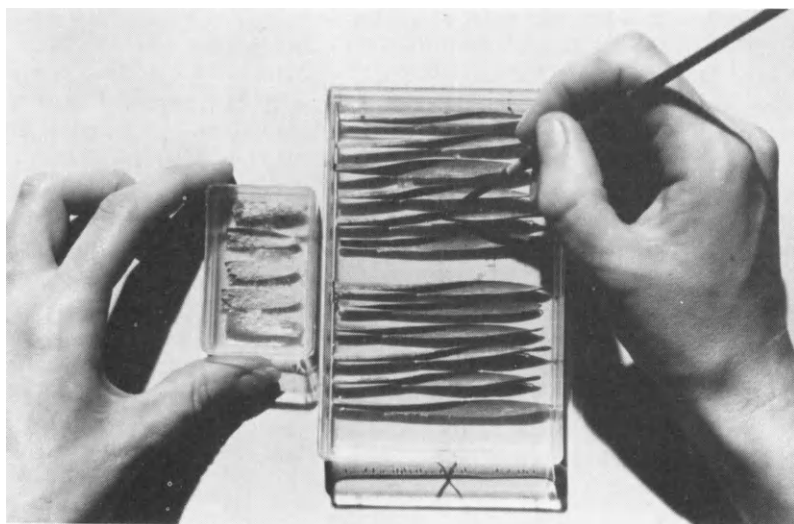


Figure 3.3 Testing for resistance to powdery mildew of barley can be carried out on segments of detached leaves supported on agar containing 150 ppm benzimidazole. Conidia are transferred from infected leaves on a soft paintbrush and the inoculated leaves are incubated in polystyrene boxes with tight-fitting lids. (By courtesy of Dr M. S. Wolfe, Plant Breeding Institute, Cambridge)

supported on agar containing 150 ppm benzimidazole in plastic boxes with tight-fitting lids (*Figure 3.3*).

NATURAL AND INDUCED FIELD EPIDEMICS

Plant breeders may be called upon to breed for resistance to two main groups of epidemic fungal diseases. The first group contains those which are damaging in most years and the second those which cause damage only sporadically. Powdery mildew of barley in the UK falls into the first group. The second group includes late blight of potato, yellow (stripe) rust of wheat and downy mildew of sugar beet, which cause serious losses in some years only, even in susceptible varieties. The main factors responsible for the infrequency of such attacks are (1) the absence of suitable environmental conditions for the development of disease; (2) insufficient inoculum to start an epidemic.

Differences between these two groups of epidemic diseases are relevant in several ways to the breeder. It is, for example, more difficult to assess the importance of resistance to those diseases which occur sporadically than it is to assess the importance of resistance to those which frequently cause damage. It may be unwise, however, to neglect diseases which appear to be economically unimportant. Powdery mildew of sugar beet has been present in California since 1934 but did not cause severe damage until 1974 and 1975. During the interim period there was no testing for resistance to this fungus, with the result that the varieties grown there in recent years are very susceptible to the disease (Kontaxis, 1976).

It is difficult to carry out an effective programme of screening for resistance to sporadic diseases if one relies solely on natural disease epidemics, for unfavourable environmental conditions might mean the loss of years of selection and breeding. With such diseases it is necessary to encourage the development of natural epidemics, to start an artificial epidemic, or to select for resistance in an area where natural outbreaks occur. All three strategies have been adopted in breeding for resistance in many crops. Natural epidemics of some diseases can be encouraged by artificially raising the relative humidity of the air around test plants, or by wetting the leaves by means of overhead sprays or other forms of irrigation. This latter method has been used extensively in testing for resistance to late blight of potatoes and to *Septoria* leaf spot in wheat. Artificially induced epidemics can be started by inoculating all, or a proportion of, the test plants or by inoculating plots of very susceptible varieties which are distributed evenly among the plots of breeding material. Sugar beet downy mildew, caused by *Peronospora farinosa*, is widespread only in humid conditions and is common only in certain years in the drier areas of eastern England. To facilitate testing for resistance to this disease, sugar beet breeders in the UK often test their breeding material in Wales where humid conditions usually prevail; sugar beet is not cultivated in Wales and it is therefore necessary to introduce inoculum into the selection plots. This is done by inoculating rows of susceptible sugar beet varieties with conidia of *P. farinosa* at the beginning of the growing season (Byford, 1969).

It is comparatively easy to exploit natural epidemics in selecting for resistance to diseases which occur regularly. Under these conditions it is difficult, however, to maintain disease-free control plots and this impedes assessment of the yield losses caused by disease in different varieties. Frequent fungicide treatments can be applied to control plots in attempts to keep them free from disease, but this may be of no avail where there is a high density of air-borne inoculum. Fungicides may also produce side effects on the control plots thus giving rise to spurious and misleading yield differences between varieties. Ideally, breeding material should be tested under natural conditions of infection, and also in artificially inoculated plots with corresponding uninoculated, disease-free plots. Such a combination of testing methods would ensure that there was not complete dependence on the presence of suitable environmental conditions for natural epidemics, and would give the maximum amount of information about different types of resistance which might be present in breeding material.

Field epidemics of a disease, employing natural spread of pathogens, can be encouraged in areas where the disease is endemic or where the environment might be expected to favour its spread to experimental plots from inoculated infection foci. The experimental plots must be laid out in an appropriate experimental design (e.g. in randomized blocks) so that each plant has an approximately equal chance of becoming infected. The severity of disease on each plant or group of plants can be assessed at intervals throughout the growing season using the methods described on page 32. The position of individual plants or plots that show little disease on any one inspection date can be marked with a cane or a label so that they can easily be identified on subsequent occasions and at harvest. This system allows promising plants or plots to be earmarked for selection purposes. Alternatively, badly infected plants can be removed at intervals throughout the season so that only the more resistant plants remain to be harvested. However, this has the disadvantage of continuously decreasing the

amount of inoculum which is available for testing the resistance of remaining plants. Where plants are to be selected for tolerance, it is necessary to obtain yield data from plots irrespective of the severity of disease symptoms.

TESTS USING ARTIFICIAL INOCULATION

Inoculation methods

It is still possible to select for resistance to a disease in the field, even in the absence of natural or induced epidemics, by inoculating whole plots or individual test plants with the causal fungus. Where environmental conditions are unlikely to favour natural spread of the disease, suitable conditions for inoculating with the pathogen and for its subsequent development must be provided. These conditions can be achieved in several ways. For example, *Streptomyces scabies*, which causes powdery scab in potatoes, will infect tubers in dry soil at a certain stage only and testing for resistance to this disease in the field is often hampered when there is heavy rainfall at the crucial stage of tuber growth. Dry soil conditions can be ensured, however, when the soil containing the test plants is protected from rainfall. At the Plant Breeding Institute, Cambridge, plots of potatoes are



Figure 3.4 Testing for resistance to diseases requiring humid conditions can be carried out in polyethylene greenhouses, which can be ventilated or cooled by fans or air-conditioners. (By courtesy of Dr K. Doodson and Dr R. Priestley, National Institute of Agricultural Botany, Cambridge)

grown in soil that is known to be infected with *S. scabies*, and these plots are covered with a transparent plastic 'tunnel' supported on a metal framework. This provides very suitable conditions for the development of powdery scab and for comparing the resistance of potato clones to the disease (Jellis, 1974). Such tunnels can also be used in testing for resistance to other diseases (*Figure 3.4*).

Many fungal pathogens of leaves require a saturated atmosphere or free water for germination of spores or subsequent penetration, and these conditions can be achieved by tightly enclosing the test plants or groups of plants in plastic bags after inoculation. The humidity of the air inside the bags will then usually increase to a sufficient level because of transpiration from the leaves. Condensation of water on the surface of the leaves inside the bag can be encouraged by lowering



*Figure 3.5 Small plots of adult wheat plants can be inoculated with *Septoria nodorum* by spraying them with a suspension of spores in water. The plots are covered with a polyethylene bag for 24 hours to maintain a high humidity around the inoculated leaves. (By courtesy of Dr P. R. Scott, Plant Breeding Institute, Cambridge)*

the temperature of the air surrounding the sealed bags. The humidity can also be increased by spraying the test plants with a fine mist of water either before or after inoculation, or by inoculating using a suspension of spores in water (*Figure 3.5*).

Where field plants are too small to be enclosed tightly in a plastic bag, they can be covered with flower pots after inoculation, to maintain the high humidity which is often essential for spore germination. The bags or pots are removed after an appropriate interval, the length of which will depend on the pathogen concerned. In inoculating wheat plants with *Puccinia striiformis* the author has found that most infections occur if inoculated plants are covered for 48 hours after inoculation, provided that cloudy conditions prevail. In hot, sunny conditions, however, the air temperature inside the cover may rise to such an extent that the pathogen is killed and the incubation period should be curtailed in these circumstances. Under these conditions the use of opaque or black plastic bags may be helpful because, in the sun, the temperature inside them is generally less than it is in transparent plastic bags. On the other hand, both the host plant and the pathogen may suffer if light is excluded for too long.

Selection for resistance to soil-borne diseases is usually carried out in soil that is known to harbour the causal pathogen. These diseases can often be encouraged by the continuous cropping of susceptible host plant species in the same soil, and uniform testing conditions can easily be obtained in this way. Where this procedure is not possible or desirable, inoculum of most pathogens can be prepared in the laboratory and incorporated in the soil of the field where screening tests are to take place.

Preparation, storage and application of inoculum

Inoculum of leaf-attacking fungal pathogens usually consists of spores, which may be accompanied by pieces of mycelium, and is applied either as a dry dust or as a suspension in water or oil. Powdered talc is often used as a dry diluent

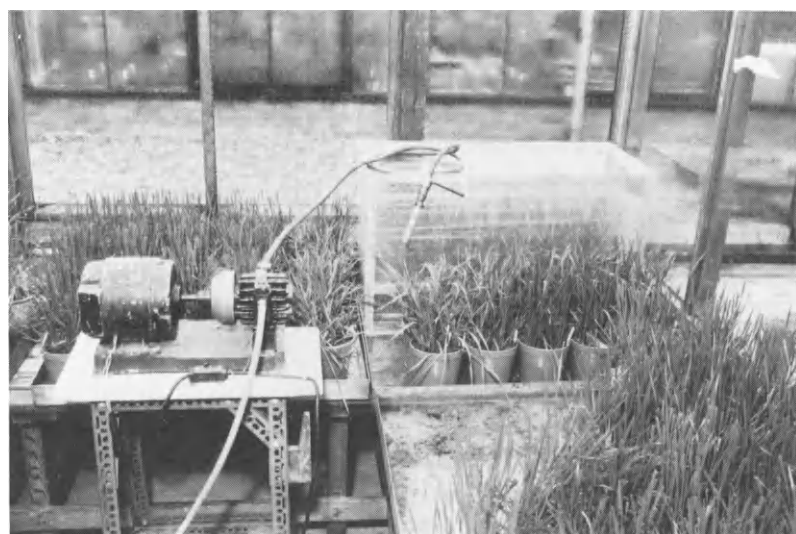


Figure 3.6 Apparatus for inoculating wheat with *Puccinia striiformis* in the glasshouse. Uredospores mixed with sterile talc are distributed by compressed air over the plants which are then enclosed for at least 24 hours in a sealed plastic cage to maintain high humidity to encourage spore germination. (By courtesy of Dr K. Doodson and Dr R. Priestley, National Institute of Agricultural Botany, Cambridge)

in an attempt to obtain a more even distribution of spores on the leaves of the test plants. Many different pieces of apparatus have been used to apply inoculum as dusts, ranging from camel-hair paintbrushes, or soft plastic bottles which are squeezed so that the powder is puffed or shaken out over the test plant, to very



Figure 3.7 Leaves of plants can be inoculated uniformly with fungal spores in a settling tower. The spores are introduced into the top of the tower and are allowed to settle on the plants inside. (By courtesy of Plant Breeding Institute, Cambridge)

sophisticated settling towers (*Figures 3.6, 3.7, 3.8 and 3.9*). Large-scale inoculations can be carried out using high-pressure jets of air to expel spores from special containers over the plots to be inoculated. Spores of certain pathogens, for example conidia of powdery mildews, are easily damaged by methods which can be used for rust fungi. With such delicate pathogens, leaves of infected plants can be lightly shaken over the plants to be inoculated so that the spores are gently dislodged and fall on to the plants beneath. Although this method does not result in a very even distribution of spores on the test plants, it is often more effective than other inoculation methods. Where only a few plants have to

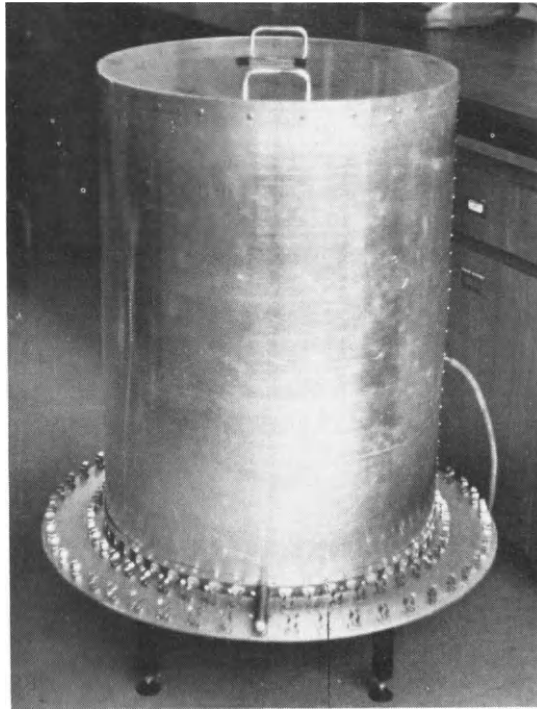
be inoculated, delicate spores can be brushed on to the leaves by means of a soft paint brush.

Successful inoculation of many pathogens can be achieved only if dry spores are used, because the spores are damaged quickly if they become too wet. Spores of many rust and powdery mildew fungi fall into this category. There are many advantages in using a liquid suspension of spores, however, and light mineral oil has been used successfully for suspending uredospores of certain rusts, before inoculation.

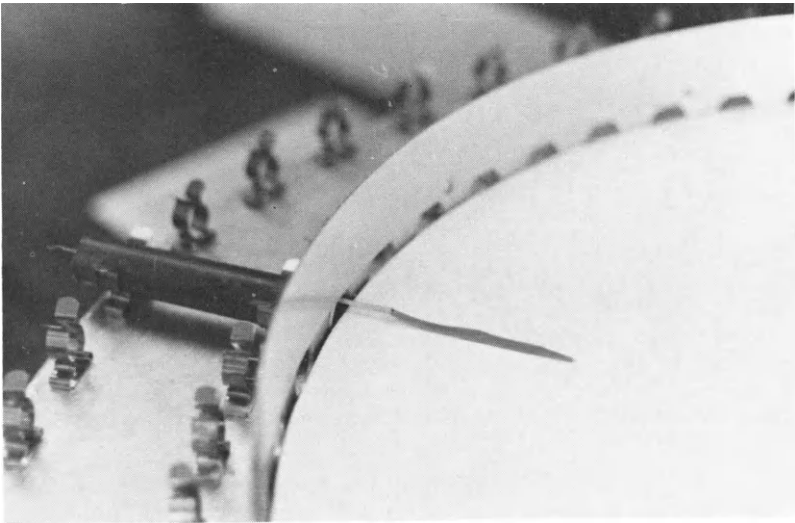


Figure 3.8 Plants can be placed on a slowly revolving platform at the base of the settling tower to increase the uniformity of spore deposition on the leaves. (By courtesy of Plant Breeding Institute, Cambridge)

The spores of many other pathogens are not damaged by being immersed in water and with these, spraying test plants with a suspension of spores in water is usually the easiest and most effective method of inoculation. This is particularly true of most Phycomycetes, the zoospores of which are generally disseminated in water, and of pathogens, the spores of which are usually splash-dispersed by raindrops in the field. Spore suspensions of many fungal pathogens can be prepared by mixing a culture medium of sterile wheat kernels, containing the mycelium or spores of pathogens, with water to obtain a suitable consistency for spraying. This method has been used for inoculating wheat with *Septoria nodorum* (Brönnimann, 1968; Scott and Hollins, 1974) as shown in *Figure 3.5*. Inoculum of some fungi can also be prepared by washing spores and mycelium directly from the surface of leaves from an infected host plant by gently shaking



(a)



(b)

Figure 3.9 Assessment of sporulation of rust fungi on wheat seedlings. (a) Leaves of wheat seedlings can be uniformly inoculated with spores of fungi in a small settling tower. (b) Uredospores are injected into the tower by compressed air and are allowed to settle on the leaves which are laid horizontally on a fixed platform. The seedlings are grown in tubes which are clipped horizontally to the perimeter of the platform. After inoculation the seedlings are removed and grown on with individual leaves in glass tubes to assess the amount of sporulation (see Figure 3.2). (By courtesy of Dr R. Johnson, Plant Breeding Institute, Cambridge)

them with water in a suitable container for a few minutes. Damage to the host tissues must be avoided during this process, because plant extracts can inhibit spore germination and the subsequent growth of some fungal pathogens, as in the case of *Peronospora farinosa* which is inhibited by sap from sugar beet leaves (Russell and Evans, 1968).

Suspensions of fungal spores and mycelium in a liquid are usually applied to test plants by a pressure-sprayer which can be operated by hand or machine; in so doing, delicate spores must not be subjected to very high pressure. Spore suspensions can also be applied by means of a hypodermic syringe (*Figure 3.10*).



Figure 3.10 Wheat flowers can be inoculated with a suspension of spores of the loose smut fungus (*Ustilago tritici*) by means of a hypodermic syringe. (By courtesy of Dr K. Doodson and Dr R. Priestley, National Institute of Agricultural Botany, Cambridge)

The type and quality of water in which the spores are washed or suspended can also be important. Tap water may contain chlorine, fluorides and other impurities, which can be fungitoxic and can therefore interfere with spore germination (Russell and Evans, 1968). It is usually preferable, therefore, to use deionized or rain water in preparing inoculum of fungi.

Spores of some pathogens, such as those of the rust fungi, are fairly robust. Such spores can be collected using a 'cyclone-head' collector, which is attached to a vacuum pump that sucks spores from the surface of infected leaves into a glass or plastic tube (*Figure 3.11*). Many pumps are reversible so that the

collected spores can be blown out of the tube again to inoculate breeding material. This method of inoculation, which is more appropriate to the glass-house and laboratory than the field, can give a very even distribution on the leaves of test plants.



Figure 3.11 Uredospores of rust fungi can be collected from infected leaves by a cyclone head attached to a vacuum pump. (By courtesy of Dr K. Doodson and Dr R. Priestley, National Institute of Agricultural Botany, Cambridge)

Spores of several fungi are so strong that they can be stored under suitable conditions for periods ranging from several weeks to more than two years. For example, uredospores of rust fungi can be freeze-dried and stored in glass vacuum-sealed ampoules (Figure 3.12). Uredospores which are stored in this way at 0–5°C can retain their viability for several months. Alternatively, uredospores can be stored in plastic sachets or gelatin capsules in a liquid nitrogen refrigerator and these can remain viable for many months. Such spores will germinate satisfactorily only if they are hydrated by placing the unsealed sachets in a warm water bath at about 20°C for about two minutes after being removed from the refrigerator. It should be realized that uredospores of rusts are unusually resistant to damage, whereas inoculum of most fungal pathogens cannot be stored for long periods. It is usually necessary, therefore, to maintain most fungal pathogens in culture or on living infected plants in order to provide a regular supply of inoculum for use in breeding programmes.

Certain fungi have resting spores or other propagules that are even tougher than the uredospores of rust fungi, and these can be stored from season to season without special facilities. For example, spores of *Tilletia caries*, which causes bunt in wheat, are protected in bunt balls that can be stored in cool, dry conditions for long periods. Seed from healthy plants can be inoculated with bunt spores by mixing the seed with the spores that are released from crushed bunt balls. Sclerotia, which are tough resting bodies composed of mycelium, are produced by several fungal pathogens, for example the clover rot fungus



Figure 3.12 Uredospores of rust fungi can be preserved for many months after freeze-drying in glass ampoules. This photograph shows such ampoules being sealed before they are removed from the freeze-drier for storage. (By courtesy of Dr K. Doodson and Dr R. Priestley, National Institute of Agricultural Botany, Cambridge)

Sclerotinia trifoliorum. Such resting bodies are extremely resistant to adverse environmental conditions and can therefore easily be stored for long periods until they are required for inoculation. Under suitable conditions, particularly high atmospheric humidity, the sclerotia can be encouraged to germinate and to produce spores which start new infections.

With soil-borne diseases, and those which attack the base of plants, it is usually necessary to incorporate inoculum into the soil or to apply it to the soil surface. In breeding for resistance to *Cercospora herpotrichoides*, the cereal eyespot fungus, short pieces of dead wheat straw that have been infected with the pathogen in the laboratory are planted in the soil beside field plants that are to be tested for resistance (Lupton and Macer, 1955; Scott and Hollins, 1974). This pathogen, which can live saprophytically in the dead straw, spreads to the adjacent test plants by means of ascospores which develop on the surface

of the straw. In selecting for resistance to root diseases, such as take-all of wheat caused by *Gaeumannomyces (Ophiobolus) graminis*, inoculum is prepared in the laboratory and is either mixed with the seed before drilling or with the soil in which testing for resistance is to be carried out. The methods employed by Scott (1971) usually involved inoculum prepared from a pure culture of *G. graminis* growing on a mixture of sand and maize-meal.

TESTS USING TOXINS

Screening for resistance to some necrotrophic fungi can be carried out in the absence of the pathogens concerned. These fungi and many plant pathogenic bacteria produce toxins which kill the tissues of the host plant (Strobel, Hess and Steiner, 1972; Strobel, 1976). Toxins of several pathogens, including *Helminthosporium sacchari* (leaf blight of sugar cane) and *H. victoriae* (leaf blight of oats), have been isolated and used to screen plants for resistance (Luke and Wheeler, 1964; Steiner and Byther, 1969). For example, a specific toxin from *Periconia circinata* has been applied to sorghum breeding material in mass screening tests for resistance to milo disease (Schertz and Tai, 1969).

Tests involving toxins, which cause the same kind of symptoms as do natural infections of the pathogen concerned, are much easier to carry out than are normal screening tests. Selection for tolerance to the toxins of several pathogens has been very effective but such tolerance is not necessarily associated with resistance to the pathogen itself. For this reason, some tests involving natural or artificial inoculation with the toxin-producing pathogens should be carried out to supplement testing procedures using artificial applications of toxins.

Disease Assessment Methods

The plant breeder can identify disease-escape tendencies by inoculating his breeding material and recording the percentage of plants in each line or genotype which becomes infected. Alternatively, the average number of separate infections on each inoculated plant or on a particular part of each plant can also be used to compare the degree of susceptibility to inoculation of individual plants.

The growth rate of a foliar pathogen on different host plants can usually be compared by measuring the dimensions of a random sample of the colonies which develop on them. For example, the length of yellow stripes on inoculated leaves gives a good indication of the relative resistance to yellow rust of different wheat varieties (Priestley and Doling, 1976). Scott and Hollins (1974) have compared levels of resistance to eyespot (*Cercospora herpotrichoides*) in wheat by recording the size of eyespot lesions at the base of the stem of infected plants; lesions girdle the stems completely in very susceptible plants whereas they are absent or very small in resistant plants.

The extent of growth and sporulation of a pathogen on the leaves or stems can be estimated by eye with the aid of a diagrammatic key, which is usually based on the proportion of the total area that is diseased (Figure 3.13). Other types of visual scoring systems have also been developed, some of which are extensively used (e.g. Peterson, Campbell and Hannah, 1948; Saari and Prescott, 1975). For example, in comparing the amount of sporulation of *Puccinia*

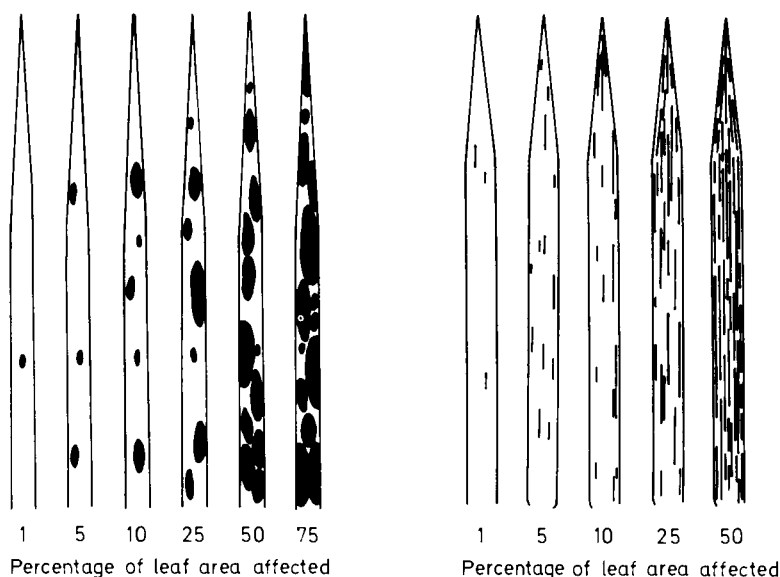


Figure 3.13 Keys for estimating the proportions of diseased areas on infected wheat and barley leaves. (a) powdery mildew (*Erysiphe graminis*). (b) yellow (stripe) rust (*Puccinia striiformis*)

striiformis on leaves of different wheat genotypes by eye, Russell and Hudson (1973) used the following 0–5 scale:

0 = no sporulation;

1 = isolated uredosori on less than 25% of leaves;

2 = isolated uredosori on more than 25% of leaves;

3 = uredosori in short stripes and many isolated uredosori;

4 = profuse sporulation on 25–50% of leaves;

5 = profuse sporulation on more than 50% of leaves.

Arbitrary scales ranging from 0 (no symptoms) to 5 or 10 (very severe symptoms) are often used and these can give very reproducible results when used by experienced and competent observers. The main advantage of such scoring systems is that they are rapid, with an acceptable degree of accuracy, and are usually quite adequate for ranking a series of test plants or plots in order of increasing severity of symptoms.

Plant breeders have recognized several reaction types shown by the host in response to infection with foliar pathogens. For example, five reaction types to rust fungi have been distinguished in wheat (Figure 3.14). The reaction types which represent the most resistant responses are those with little or no chlorosis, i.e. reaction types 0, 1 and 2. Plants showing type 3 and 4 reactions have usually been discarded as being too susceptible. The reaction type method of disease assessment is mainly a measure of the effectiveness of hypersensitivity in controlling a pathogen, and varieties that have been selected on the basis of a resistant reaction type alone have often shown a very race-specific interaction with the pathogen concerned. It would seem unwise, therefore, to restrict disease assessment to a single method, such as the recording of reaction type. Separate assessments of the effectiveness of several different types of resistance, including disease-escape tendencies, the degree of inhibition of growth and

sporulation of the pathogen, and the length of the generation time of the pathogen, are likely to be much more useful than merely recording the reaction type.

With root diseases, such as take-all of cereals, direct assessment of damage caused by infection is often difficult to achieve without destructive sampling of the test plants. In later generations of selection and testing, where such sampling may be possible, the severity of symptoms on roots of individual plants can be

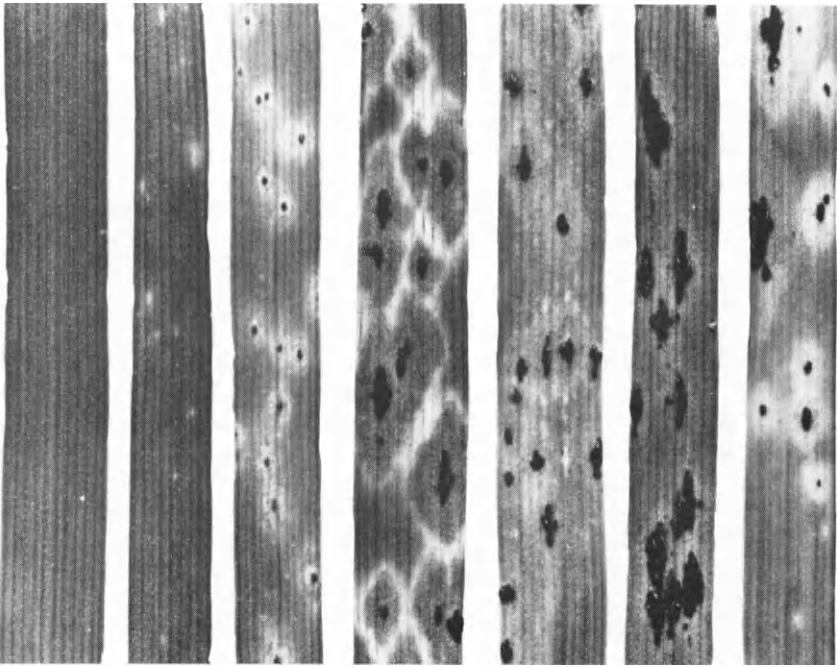


Figure 3.14 Infection types which can be used to assess resistance of wheat to stem rust (*Puccinia graminis* f.sp. *tritici*) and other rust diseases of cereals. From left to right:— 0 (no macroscopic symptoms); 1 (small pustules with underlying necrosis); 2 (larger pustules with underlying necrosis); 3 (large pustules with little necrosis); 4 (compatible reaction with very large pustules and very little necrosis); X (mesothetic reaction involving a range of pustule sizes). (By courtesy of Dr R. McIntosh, University of Sydney, Australia)

compared. Possible methods include visual assessment of the extent of root discoloration or of the proportion of the root system that is diseased. Where destructive sampling is not possible, the breeder must normally look at parameters other than root growth, including the growth rate of the aerial parts of infected plants, or the development of secondary symptoms. For example, the extent of damage to the roots of wheat by *Gaeumannomyces* (*Ophiobolus*) *graminis*, the cause of take-all, is reflected in the growth rates of development of stem and leaves and in the number of barren ears (whiteheads) that are produced. The relative resistance to take-all of different wheat genotypes can therefore be assessed by comparing the vigour of the leaves and stems, by counting the number of whiteheads, and by comparing the yields of grain.

Perhaps a more accurate method of comparing the growth rates of a pathogen on different host plants is to measure the amount of fungal chitin that is produced in a particular period. Ride and Drysdale (1972) have developed a rapid technique for estimating the quantity of chitin in plant material using an assay which does not require very sophisticated apparatus or great chemical expertise. This technique estimates the amount of fungal cell wall present, whether the mycelium is wholly inside the host plant or predominantly outside (see also page 33).

Factors Affecting the Expression of Resistance

Plants that are resistant to a disease under one set of conditions are not necessarily equally resistant under other conditions. Many factors can alter the expression of inherited resistance.

Where resistance to a disease is race-specific, a host plant may show few, if any, disease symptoms in the presence of incompatible variants of the causal pathogen, but may become severely diseased when attacked by compatible variants. Frequently it has been reported that certain crop plant varieties are resistant to a certain disease when grown in some parts of the world, but are susceptible in others. Such a situation usually indicates that the resistance concerned is race-specific, and the breeder should then beware of using such resistance in his breeding programmes. Although compatible variants of the pathogen may not have been recorded in the areas in which his varieties may be grown, such variants will probably become widespread when these varieties are exposed to the pathogen on a large scale. It is useful, therefore, for breeding material to be exposed to as many pathogen variants as possible so that material with race-specific resistance to diseases can be discarded. Many organizations, for example the International Wheat and Maize Improvement Center (CIMMYT), conduct extensive trials of wheat genotypes in many parts of the world where attacks of the main cereal diseases can be expected. In this way, very valuable information can be collected concerning the potential durability of disease resistance sources in wheat, because they are exposed to a broad spectrum of pathogen variants.

The expression of resistance to a disease can also be affected by the age of the host plant. Adult plants are usually more resistant to fungal diseases than are seedlings or juvenile plants. For example, it is a general rule that wheat plants become more resistant to the same isolates of *Puccinia striiformis* as they grow older, the difference in degree of resistance to yellow rust between seedling and adult plant being much greater in some wheat varieties than others. Russell and Hudson (1974) reported that two wheat varieties, Little Joss and Nord Desprez, are equally susceptible to yellow rust as seedlings, but that adult plants of Little Joss are much more resistant to the disease than are those of Nord Desprez. Some of the mechanisms which are responsible for the differences in resistance of these varieties to yellow rust have been characterized (Russell, 1976). These include a reduced rate of growth and sporulation and an extended generation time of *P. striiformis* on Little Joss.

Other examples of adult-plant resistance to fungi are found in barley with powdery mildew and in sugar beet with downy mildew. Barley seedlings are usually more susceptible to powdery mildew, caused by *Erysiphe graminis*,

than are corresponding older plants. This effect of age on the expression of resistance is greater in some barley varieties than others and is caused by a combination of resistance factors, including reduced germination of *E. graminis* conidia, fewer penetrations of the host and a slower growth rate of the pathogen and decreased sporulation (Russell, Andrews and Bishop, 1975; 1976). Sugar beet seedlings of some varieties and breeding lines are very susceptible to downy mildew, caused by *Peronospora farinosa*, whereas they soon become much more resistant to the disease (Russell, 1972). The cotyledons and the first pair of true leaves of sugar beet are particularly susceptible to downy mildew, and these differ morphologically and physiologically from the leaves which are subsequently produced; the third and fourth leaves are intermediate in morphology, physiology and disease resistance between the first two leaves and the 'adult' leaves (Russell and Barford, 1971).

On the other hand, young plants are more resistant than older plants to some diseases, for example powdery mildew of wheat (*Erysiphe graminis*). This phenomenon of 'adult-plant susceptibility' is much less common than adult-plant resistance and very little is known about the mechanisms responsible.

Major resistance genes, particularly those which control hypersensitivity, are usually equally effective at all stages of the growth of the host plant and are less affected by environmental changes than other kinds of resistance. A notable exception to this general rule is resistance to wheat stem rust (*Puccinia graminis*) which is conditioned by the *Sr₆* gene. This gene confers resistance to *P. graminis* at low, but not at high, ambient temperatures; the expression of resistance which is controlled by the *Sr₆* gene will, therefore, depend on the temperature of the air surrounding the inoculated plants. Although there have been few reports of a similar temperature sensitivity of resistance genes with other diseases, further investigation may show that this is not uncommon. Such sensitivity could partly explain those differences in the expression of resistance which have been encountered with certain genotypes when tested under very different environmental conditions.

The nutrition of the host plant can also affect the expression of resistance to diseases. Heavy applications of nitrogenous fertilizers usually predispose plants to infection by fungal pathogens; for example, an excess of nitrogenous fertilizers generally increases the susceptibility of barley to powdery mildew (Last, 1957). The effects of altered host-plant nutrition on disease susceptibility can vary greatly in different genotypes of wheat and barley (Russell and Hudson, 1973) and of sugar beet (Russell and Evans, 1972). Additional applications of certain nutrients, including potassium chloride, sodium chloride and boric acid can significantly decrease the severity of yellow rust (*Puccinia striiformis*) in some winter wheat varieties but not in others. Thus, there often seems to be a very specific interaction between the expression of disease resistance in particular host genotypes, and nutrient treatments. This may explain in part why a host genotype or breeding line may seem to be more resistant to a disease in some field trials than in others. This lability of expression of resistance to fungal diseases in different environments is rarely encountered with resistance that is controlled by major genes, and particularly that which involves hypersensitivity. Such lability may help plant breeders to identify those types of resistance to a disease which are not based on major-gene hypersensitivity so that they can, if they wish, concentrate their efforts on types which may be more durable than hypersensitive resistance.

The Control of Fungal Disease by Resistant Varieties

Inherited resistance to fungal diseases should be the mainstay of control measures. Attention has often been focussed on the so-called failures of resistant varieties to control diseases effectively, following attacks by resistance-breaking races of the causal pathogen. These failures are particularly conspicuous because they are seen against a background of successful control of diseases by resistant varieties. The failures are very much an exception to the general rule of success.

Much of this success in the past has resulted from almost unintentional selection for resistance to disease by plant breeders. It has been common practice for breeders to discard from their breeding programme plants which are exceptionally susceptible to any disease. This has resulted in a gradual but significant improvement in disease resistance over the years in most of the important agricultural crops of the world. The degree of resistance which has been achieved in this way has not been spectacular or complete, but a complete control of disease is not necessary and may not even be desirable. Adequate control has been achieved if damage by a disease does not become economically significant and if the disease does not present a threat to other crops. Disease control should not, therefore, be equated with no disease. Many of the problems that have been associated with breakdowns in resistance have followed attempts by plant breeders to achieve complete control of a disease under all conditions. Two examples will help to illustrate this point.

Following the catastrophic outbreak of late blight of potatoes (*Phytophthora infestans*) in western Europe in the middle of the nineteenth century, plant breeders discarded blight-susceptible plants from their stocks, and several varieties with an acceptable level of field resistance to the disease were produced as a result. These varieties included Majestic, which was the most widely grown maincrop variety in the UK for more than sixty years after its introduction in the early 1900s. This variety owed much of its popularity to the fact that the tubers were very resistant to blight. Although blight did occur in seasons which favoured the spread and development of the disease, there was no recurrence of the severe blight epidemics of the mid-nineteenth century. Even in the so-called blight years, the disease could usually be controlled to some extent on this variety by spraying with appropriate fungicides. New problems with late-blight control were encountered, however, when plant breeders introduced major-gene resistance from *Solanum demissum* and other species of *Solanum* into cultivated potatoes (see page 117). These major resistance genes, which control a hypersensitive response to *Phytophthora infestans*, were soon matched by virulence genes in the pathogen so that the new varieties soon became badly diseased. As a consequence, there has been a renewed interest in field resistance to blight which gives a partial, but often adequate, control of the disease in the field. The problems that were encountered with resistance-breaking races of *P. infestans* in the 1940s and 1950s were clearly the direct result of the use of major resistance genes (controlling a hypersensitive reaction) introduced from alien species.

In sugar beet, several fungal diseases of the leaves, including downy mildew (*Peronospora farinosa*), powdery mildew (*Erysiphe betae*) and rust (*Uromyces betae*), have not usually caused severe losses of yield in the UK even when conditions have favoured their development and spread. This happy situation seems primarily to be attributable to an adequate level of field resistance to

these diseases in most sugar beet varieties. Field resistance to each of these diseases seems to be complex, involving many different kinds of mechanism, and is controlled polygenically. Over the years, plant breeders have discarded severely diseased plants, thereby ensuring an adequate level of disease resistance. No breakdowns of this field resistance, caused by attacks of resistance-breaking forms of the causal pathogens, have been reported and the resistance appears to be non-race-specific. Sugar beet lines that are bred from severely diseased plants are themselves often very susceptible to the disease or diseases concerned. This supports the contention that these diseases have been kept in check largely by mass selection of field-resistant plants. Certain sugar beet varieties in the USA have been badly attacked by powdery mildew, presumably because they were bred under conditions which did not allow selection for field resistance.

As in potatoes, however, there is a danger that the advantage of durable field resistance could be lost in sugar beet if attempts were made to introduce certain other types of resistance. Russell (1969) discovered, in an inbred line of sugar beet, a hypersensitive form of resistance to downy mildew that is controlled by a single dominant gene. Within a comparatively short time, variants of *P. farinosa* were found which could attack this particular inbred line. If this resistance had been used in the production of new sugar beet varieties, the complex field resistance to downy mildew that is present in most sugar beet might have been lost, because it would have been masked by hypersensitivity. A serious breakdown in resistance to downy mildew might then have occurred in these varieties.

These examples emphasize some very important general rules in breeding for resistance to fungal diseases. First, it is usually possible to obtain, by empirical selection methods, a level of field resistance which will give satisfactory control of a particular disease under most conditions. Such field resistance will probably be complex and therefore more difficult to manage in a breeding programme than major-gene resistance, but this disadvantage should be counterbalanced by a greater degree of durability.

Second, major-gene resistance, particularly if it involves hypersensitivity, is often race-specific. The potential disadvantages of using such resistance should be carefully considered because the presence of major resistance genes can seriously complicate subsequent selection for partial field resistance. For example, polygenically controlled 'partial' resistance to *Puccinia hordei*, expressed in several barley varieties as an extended generation time of the pathogen, can be completely masked in the presence of major resistance genes (Parlevliet and Kuiper, 1977). Similarly, the existence in potatoes of major *R* genes for resistance to late blight in breeding material made it difficult to select for field resistance, or to test for its presence. In the absence of positive selection for field resistance, the level of field resistance quickly became eroded in varieties with major-gene resistance; in consequence, these varieties became severely diseased when they were attacked by resistance-breaking races of *Phytophthora infestans*. This loss of background field resistance was called the 'Vertifolia effect' by Van Der Plank (1963). Breeders of winter wheat have experienced similar difficulties in trying to select for non-hypersensitive types of resistance to yellow rust (*Puccinia striiformis*) in the presence of major resistance genes which can mask the expression of other kinds of resistance. In an attempt to overcome this difficulty, some wheat breeders have

retained only susceptible seedlings in the F_2 and succeeding generations, on the assumption that such plants do not carry major resistance genes. The susceptibility of the selected plants at the adult-plant stage is then assessed and compared, and the more resistant plants are retained for breeding (Bingham, 1975). Several varieties of wheat have been produced in this way, but there is no guarantee that their resistance to yellow rust will be any more durable than the resistance of varieties that have been produced by other methods.

A third important rule is that, even in the absence of the masking effects of major genes, valuable field resistance to a disease is likely to be lost unless some positive action is taken to select for field resistance. The recent severe outbreak of powdery mildew on sugar beet varieties in California is an excellent example of the potential dangers of omitting to select for resistance to a potentially important disease. If plant breeders produce varieties of crop plants which are very susceptible to a particular disease, it is almost inevitable that this disease will become more prevalent and damaging than it was before.

References

The references cited in this chapter, together with those in Chapter 4, are listed in References—Part II, pages 140–166.

EXAMPLES OF BREEDING FOR RESISTANCE TO FUNGAL DISEASES

Wheat

YELLOW (STRIPE) RUST

Yellow rust (*Puccinia striiformis* Westend) is one of the most important diseases of bread wheat (*Triticum aestivum*, a hexaploid with $2n = 42$ chromosomes) in temperate areas of the world, particularly those with cool, maritime climates such as northern Europe and north-western America. It is also common at high altitudes in some subtropical regions, including parts of East Africa, Central and South America and the Indian subcontinent. The disease has recently been recorded in warmer and more arid areas than previously, for example Egypt, Iran and Turkey. This apparent extension of the distribution of *P. striiformis* may be due to the development of new variants of the pathogen, which are able to tolerate higher temperatures, or to the cultivation of very susceptible varieties in these areas.

Yellow rust can seriously reduce grain yields, particularly when a severe attack develops before ear emergence; such attacks can cause losses of grain yield in excess of 20 per cent in very susceptible varieties (Doling and Doodson, 1968; Manners, 1969). The disease mainly affects the leaves but the glumes can also become infected. Yield is affected primarily because the carbohydrate supply to the ear is decreased as a result of reduced rates of photosynthesis and translocation and of an increased respiration rate (Doodson, Manners and Myers, 1965) and enhanced senescence of infected tissues (Harding, Manners and Myers, 1976). Infected plants of susceptible varieties have a reduced number of florets and grains per ear and sometimes a lower weight of individual grains. Leaves of infected plants are shorter and narrower than those of healthy plants, and the dry weight of the roots of susceptible plants can be reduced by more than 75 per cent following infection. Until recently, control of yellow rust was almost exclusively by the use of resistant varieties. Effective systemic fungicides have now been developed and these will undoubtedly provide valuable additional control.

Breeding for resistance to yellow rust has been a major objective in wheat breeding programmes for many years. Biffen (1907) at Cambridge in the early 1900s was the first to investigate the inheritance of resistance. In crosses of resistant species, including *Triticum monococcum vulgare* and *T. turgidum*,

with susceptible bread wheats such as Michigan Bronze, he found that resistance to yellow rust is a simply inherited recessive character, but he was not able to associate resistance with any morphological character. In the 1930s other workers described resistance controlled by single dominant genes in the variety Chinese 166 and in *Triticum spelta* var. *album*; resistance genes from the latter variety have conferred resistance to a very wide range of *P. striiformis* isolates (Zeven, Turkensteen and Stubbs, 1968). Many other sources of resistance, which are controlled monogenically or oligogenically, have since been described (Macer, 1972). Allan and Purdy (1970) made crosses between several resistant genotypes and susceptible varieties and concluded that resistance to yellow rust is controlled by a single gene in some genotypes (e.g. PI 178383, Spaldings Prolific and Sel 63301), by two genes in Nord and Holzapfel Fruh and by three genes in others. Some of these genotypes, for example Nord and PI 178383, have at least one gene in common. Pope (1969) also studied the inheritance of resistance in a number of resistant varieties and found four main patterns of segregation for resistance: (1) highly resistant wheats with resistance controlled by dominant genes (e.g. PI 178383); (2) highly resistant varieties with multiple genes for resistance, only some of which were maintained in a backcross to a susceptible variety; (3) varieties with moderate or intermediate resistance which was controlled by either dominant or recessive single genes; (4) susceptible wheats but with genes which conferred good resistance in some of their selfed progenies.

Riley, Chapman and Johnson (1968) isolated a line from a cross between a bread wheat variety, Chinese Spring, and the resistant wild grass *Aegilops comosa*, having a single chromosome from *A. comosa* in a bread wheat genetic background. The resistance of this line is controlled by a single dominant gene from the wild species, which was designated Yr_8 to distinguish it from seven previously identified major genes (Yr_1 – Yr_7) (Lupton and Macer, 1962; Macer, 1972). Many wheat varieties carry genes for resistance to *P. striiformis* derived from rye (Stubbs, Slovenčková and Bartoš, 1977). However, some Triticales, which are hybrids between wheat and rye, are very susceptible to yellow rust (Prato *et al.*, 1976).

Most of the above examples concern resistance which can be detected easily at the seedling stage. This resistance is controlled by major genes and apparently involves a hypersensitive reaction of the host cells to infection. This type of resistance has obvious advantages to the breeder, who can easily test large numbers of seedlings and can readily exploit the resistance in breeding programmes because it is simply inherited. For these two reasons, major-gene hypersensitive types of resistance to yellow rust have been very widely used, but *P. striiformis* has produced variants or physiologic races with new or recombined genes for virulence to which many previously resistant host genotypes have proved susceptible (Howard *et al.*, 1970). This situation has led to the discarding of much potentially valuable breeding material and the withdrawal of some of the most productive varieties from agriculture. In the UK a succession of new physiologic races of *P. striiformis* has been responsible since the early 1950s for the breakdown in resistance of many new varieties. This situation has led to a search for other types of resistance that are more long-lasting or 'durable' (Johnson and Law, 1975). Some of the older English and French wheat varieties, although susceptible as seedlings to most races of *P. striiformis*, have continued to show good adult-plant resistance to yellow rust in the field for many years

(Figure 4.1). For example, Little Joss, a winter wheat variety bred at Cambridge by Biffen in the early 1900s is as resistant to yellow rust today as when he first bred it. Other examples of varieties with durable resistance are Yeoman, Browick, Holdfast, Atle and Cappelle-Desprez (Howard *et al*, 1970; Lupton and Johnson, 1970), and Česká přesívka and Dobrovická 10 (Slovenčíková, 1972).



Figure 4.1 Resistance to yellow (stripe) rust (*Puccinia striiformis*) in winter wheat. A plot of a very susceptible variety (right) is severely infected whereas an adjacent resistant variety (left) is unaffected. (By courtesy of Plant Breeding Institute, Cambridge)

Although these varieties can become severely infected when levels of inoculum are particularly high, they usually show only mild symptoms and suffer little damage from yellow rust. Little Joss, Yeoman and Holdfast were grown during the mid 1960s in 127 yellow rust race nurseries in 34 countries, where they were exposed to a wide range of physiologic races of *P. striiformis* and different environmental conditions; they showed good resistance at all sites, suggesting that resistance is non-race-specific (Stubbs, Vecht and Fuchs, 1967). The

inheritance of this resistance in Little Joss has been described as polygenically controlled by some workers (Lupton and Johnson, 1970) and as simply inherited by others (Lupton, Bingham and Wilson, 1973): this suggests that the resistance may be controlled by one or more genes of large effect, and by several minor genes. Such resistance can be satisfactorily exploited in breeding programmes provided that sufficiently high levels of rust inoculum are present to permit effective selection in the field. There is a danger, however, that some of the characteristics which have been responsible for the stability or durability of yellow rust resistance in varieties such as Little Joss may be lost during selection and breeding. Varieties developed from such a programme could, in the short term, show good resistance which might quickly be eroded by resistance-breaking races of *P. striiformis*. Until the characters which confer durability of rust resistance can be recognized, breeders should, as far as possible, use as parents only those varieties with proved stable resistance, and should carry out recurrent selection for resistance in the field, preferably in conditions where plants are subjected to a very wide range of variants of *P. striiformis*.

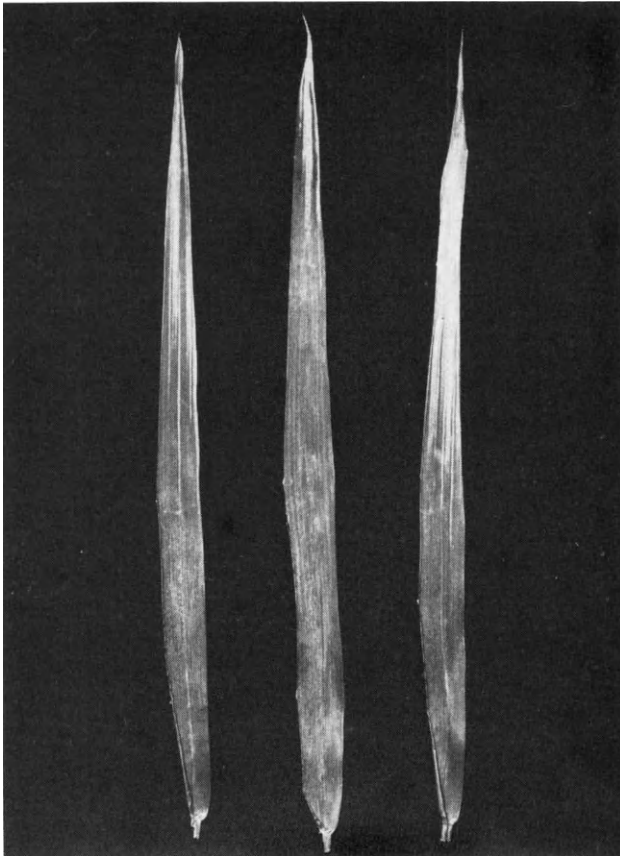
Different workers have attempted various classifications of resistance to rusts. For example, Hart (1931) recognized three main types of resistance to stem rust (*Puccinia graminis tritici*); these were protoplasmic (hypersensitive), morphologic and functional. She postulated that wheat seedlings lack effective morphologic and functional resistance mechanisms and that this is responsible for the observed differences between the resistance of seedlings and mature plants. Manners (1969) distinguished four types of resistance to yellow rust: (1) seedling resistance, in which the host is resistant both as a seedling and as a mature plant; (2) adult-plant resistance, in which the host is susceptible as a seedling but resistant as a mature plant; (3) environmentally determined resistance (commonly called 'field resistance'), in which resistance can be manifested in the seedling, in the adult plant or in both, but the expression of which is labile and is particularly sensitive to changes in temperature; (4) tolerance, in which the host is susceptible but yields well despite infection.

Many workers, including Russell and Hudson (1974) and Hermansen (1976), have confirmed that the expression of resistance to yellow rust can differ greatly at different growth stages of individual wheat genotypes. The reactions of seedlings to the disease are not, therefore, always a reliable guide to the reactions of adult plants. For this reason breeders should not confine themselves to seedling tests in selecting for resistance to *P. striiformis*. Stubbs (1968) has shown that even the first and second leaves of a wheat plant can have quite a different response to yellow rust. For example, in F_2 seedlings from a cross between Cappelle-Desprez and Michigan Amber, the first leaf was susceptible to race 8 of *P. striiformis*, whereas the second leaf was resistant. Conversely, in F_2 seedlings of a cross between Heines VII $\times$ Michigan Amber, the first leaf was resistant to race 20 but the second leaf was susceptible. It has been suggested that the reaction of the first leaf to *P. striiformis* is influenced by the endosperm, whereas that of the second leaf is dependent mainly on the genotype of the embryo. Comparative studies of the morphology and physiology of the first two leaves might, therefore, increase our knowledge concerning some of the mechanisms involved in resistance to *P. striiformis*.

Zadoks (1972) distinguished between the following two main types of resistance to cereal diseases according to the degree of specificity between the genotypes of host and pathogen: (1) differential resistance, which is equi-

valent to the 'vertical' resistance of Van Der Plank (1963), where only certain genotypes of the pathogen can cause disease on particular host genotypes; and (2) uniform (or 'horizontal') resistance, which is equally effective against all variants of a pathogen.

Types of resistance to yellow rust can also be classified in epidemiological terms, according to the nature of the resistance mechanisms concerned (Zadoks,



*Figure 4.2 Flag leaves of wheat showing differences in resistance to yellow (stripe) rust. In a susceptible variety (right) *Puccinia striiformis* has spread more than half way down the leaf from a point of inoculation near the tip. In moderately resistant (left) and resistant (centre) plants, the fungus has spread less far from the inoculation point. (By courtesy of Plant Breeding Institute, Cambridge)*

1968). This concept has been developed and extended by Russell (1976b) who has shown that different resistance mechanisms can operate in adult winter wheat plants at the following six stages in the development of *P. striiformis*: (1) **resistance to spore deposition**, where different numbers of uredospores settled on equivalent leaves of different varieties when they were exposed to a uniformly distributed shower of uredospores; (2) **resistance to spore germination** in which the germination of uredospores was inhibited on the leaf surface of some varieties; (3) **resistance to penetration** in which a smaller proportion of germinated uredospores produced germ tubes that penetrated through

the stomatal pores to produce sub-stomatal vesicles; (4) hypersensitivity, where penetrated host cells were killed; (5) **resistance to growth of the pathogen**, in which the growth of the fungus was inhibited and the extent of infection and sporulation reduced (*Figures 4.2 and 4.3*); (6) **increased generation time of the pathogen**. None of the varieties examined exhibited all six components of resistance, and the disease escape mechanisms (types 1–3) were apparently inherited quite independently from any of the post-penetration types (3–6).

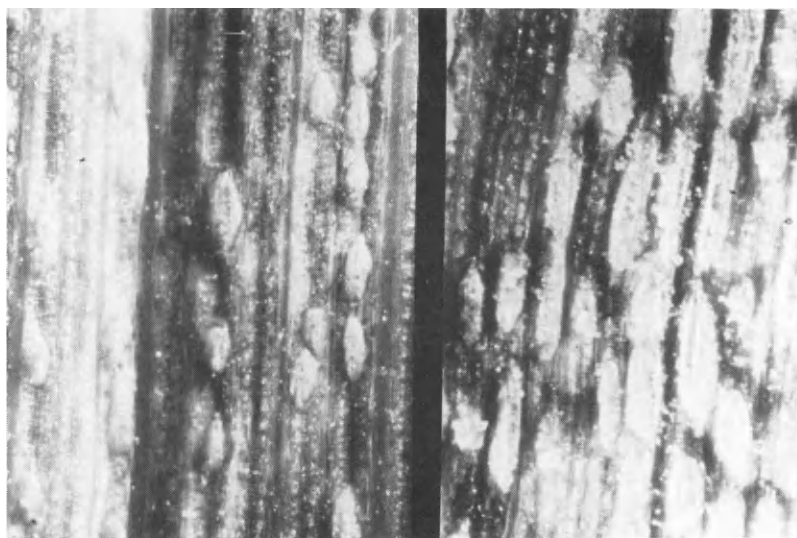


Figure 4.3 Pustules (uredosori) of Puccinia striiformis are more numerous and larger on leaves of susceptible wheat varieties (right) than on those of more resistant varieties (left). (By courtesy of Plant Breeding Institute, Cambridge)

These, and many other classifications of types of resistance to yellow rust, emphasize the many conflicting viewpoints of breeders, geneticists, and pathologists. This confused situation will remain until we have a much greater understanding of the underlying mechanisms of resistance, and of the effects of individual mechanisms on the epidemiology of yellow rust and on the evolution of new genetic forms of *P. striiformis*. The most important distinction that can be made between types of resistance at present is that some types have given a more durable control of yellow rust than others. Hypersensitivity, which is controlled by major genes, seems generally to be race-specific, particularly when it is expressed in seedlings. However, resistance that is expressed in adult plants but not in seedlings can also involve hypersensitivity (Mares and Cousen, 1977) and has sometimes been found to be race-specific. A winter wheat variety, Joss Cambier, with this type of resistance, was attacked by variants of *P. striiformis*, specially adapted to this variety, to such an extent that it became badly damaged (Johnson and Taylor, 1972). This emphasizes that adult-plant resistance is not synonymous with durable or non-race-specific resistance (Bingham, 1975). Techniques for detecting specific virulence in *P. striiformis* on adult winter wheat plants have been developed by Priestley and Doodson (1976); plants are grown in polyethylene tunnels and are inoculated as adult plants with specific variants of *P. striiformis* (*Figure 4.4*).

Successive breakdowns of resistance in many varieties with major resistance genes has focussed attention on the use of minor genes for resistance. Many wheat varieties possess minor, additive genes which confer an acceptable level of field resistance to yellow rust (Sharp and Volin, 1970). Some varieties carry major genes for resistance to certain variants of *P. striiformis*, and minor genes for resistance to other variants (Volin and Sharp, 1969). This suggests that resistance controlled by minor genes can be race-specific, and indicates in turn that polygenically controlled resistance is not necessarily non-specific. Nevertheless, there is considerable optimism that greater durability of resistance to yellow rust will result from the use of minor resistance genes in breeding programmes (Sharp, 1976; Sharp, Sally and Taylor, 1976; Stubbs, 1977).

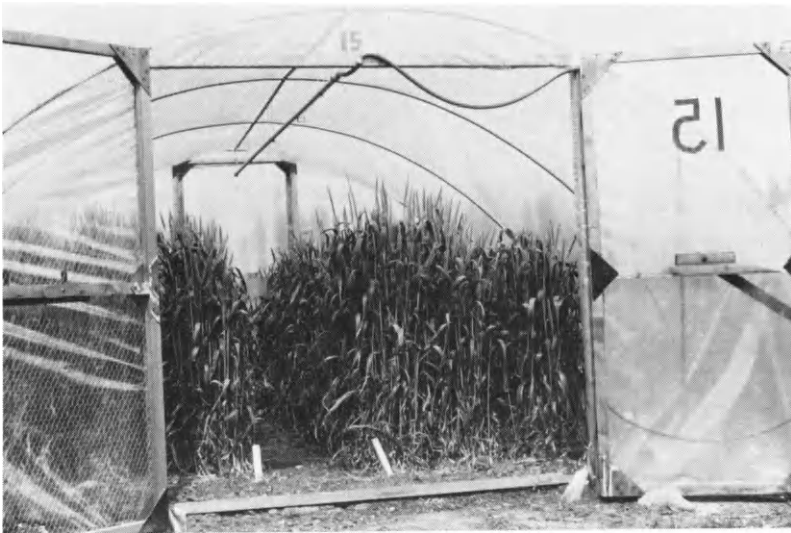


Figure 4.4 Adult wheat plants can be tested for resistance to *Puccinia striiformis* by encouraging the development of yellow (stripe) rust in polyethylene greenhouses. A high atmospheric humidity can be maintained after inoculation by means of an overhead spray irrigation system. (By courtesy of Dr K. Doodson and Dr R. Priestley, National Institute of Agricultural Botany, Cambridge)

Johnson and Law (1975) have shown that a large part of the apparently durable resistance of the variety Hybride de Bersée, and certain other French wheats, is controlled by genes on the chromosome 5BS–7BS. This chromosome has been transferred to other stocks in order to study the expression of resistance in other genetic backgrounds. This will show whether the durable resistance of Hybride de Bersée depends solely on the effect of this chromosome or on an interaction between chromosome 5BS–7BS and other genetic factors. A major difficulty, however, will be to determine whether this or any other resistance is really durable, because the demonstration of durability, by definition, depends on widespread exposure of resistance to new variants of *P. striiformis* over a period of many years.

New resistance-breaking variants of *P. striiformis* have arisen in the field in many parts of the world (e.g. Macer, 1972; Johnson and Taylor, 1972; Negulescu and Ionescu-Cojocar, 1976; Qualset, Prato and Vogt, 1977). Some of these physiologic races have been identified very soon after varieties carrying

new resistance genes have been exposed to infection: for example, the resistance of a promising new winter wheat variety, Maris Bilbo, was overcome by a race of *P. striiformis* with specific virulence for that variety while it was still undergoing small-scale yield assessment tests in England. Similarly, a new virulent race of *P. striiformis* attacked a widely grown, previously resistant variety, Pitic 62, in California in 1974, causing a very severe epidemic of yellow rust (Line, 1976).

The new variants have arisen quickly despite the absence of a known sexual phase in the life cycle of *P. striiformis*. This absence has not, therefore, limited the capacity for genetic variation in this fungus. The mechanisms involved in the origin of new races have been the subject of much speculation for many years, but it is now generally agreed that new races arise mainly by mutation or by the exchange of whole nuclei between genetically dissimilar heterokaryotic individuals. Little is known about mutation rates in *P. striiformis* but mutants, which differ from the parent types in aggressiveness, have been induced without difficulty after irradiating the fungus with X-rays. Little and Manners (1969) obtained two new races of *P. striiformis* from a mixture of two existing races on wheat leaves, suggesting that reassortment of whole nuclei had occurred between hyphae of the parent genotypes. Goddard (1976) and Taylor (1976) also each obtained a new race which combined the virulence of two parent races grown together on wheat leaves. Reassortment of whole nuclei was thought to be the most likely explanation of this phenomenon, but the possibilities of other mechanisms, such as the parasexual cycle or cytoplasmic inheritance, cannot be ruled out (Taylor, 1976). It is unlikely that the reciprocal exchange of whole chromosomes between individuals is responsible for increasing the variability of *P. striiformis* (Wright, 1976).

Future possibilities

Several proposals have been put forward regarding the more efficient use of major-gene, hypersensitive types of resistance to foliar diseases of cereals in which resistance-breaking pathogen variants have been a serious problem (Wolfe, 1972). For instance, resistance conditioned by certain combinations of major genes may be more durable than that controlled by others. Alternatively, multi-line or composite varieties, comprising near-isogenic lines which carry different resistance genes, could be developed as advocated by many workers including Borlaug (1959). In addition, the recycling of resistance genes might sometimes increase the durability of resistance controlled by major genes. It is unlikely, however, that any of these proposals would, by themselves, result in a permanent, acceptable level of resistance. The best long-term prospects for controlling yellow rust must lie in the exploitation of those types of resistance which have remained effective for many years. In the meantime, effective but costly fungicides are available to control yellow rust where previously resistant varieties are likely to be damaged by resistance-breaking variants of *Puccinia striiformis*.

STEM RUST

Stem (or black) rust (*Puccinia graminis* f.sp. *tritici*) is common in most of the major wheat-producing areas of the world and is one of the most destructive diseases of wheat. Severe epidemics of stem rust have been reported from North,

Central and South America, Australia, parts of southern and Eastern Europe, the USSR, India and Kenya. Uredospores of *P. graminis* are resistant to desiccation and to ultraviolet irradiation and can therefore survive after being wind-blown over very long distances, thus enabling the disease to spread extensively and rapidly. Wind-borne spores from Mexico are thought to have been responsible for initiating epidemics in several parts of the USA. Similarly, there is probably a passage of *P. graminis* uredospores in air currents from the western Mediterranean area to north-western Europe, including the UK, and from the Near East through the Balkans to Germany and Poland.

The potential importance of stem rust can be judged from the estimated losses of 45 million bushels and 150 million bushels of grain, in 1954 and 1955 respectively, in western Canada alone (Peterson, 1958). Control of the disease by fungicides can increase the yields of susceptible varieties in severe field infections by more than 56 per cent (Eversmeyer, Browder and Young, 1975). During the 1960s and early 1970s, however, losses from stem rust have been relatively low because of the widespread use of resistant varieties.

Resistant varieties

Although systemic fungicides, which can give a good control of stem rust, have recently been developed and can supplement other control measures, resistant varieties will probably continue to be the first line of defence against the disease. The United States Department of Agriculture (USDA) instituted a large-scale programme of breeding for resistance to stem rust as early as 1905, following an unusually destructive epidemic of the disease in the previous year. From this and other breeding programmes, many rust-resistant varieties have been produced, but most of these have given only a localized, intermittent control of stem rust. The pathogen has been able to produce new physiologic races capable of attacking one previously resistant variety after another (Stakman and Christensen, 1960).

One of the earliest resistant varieties to be produced in the USA was Kanred, a hard red winter wheat, which was first distributed in Kansas in 1917. At first it appeared to be immune to stem rust, but was soon observed to be very susceptible to certain isolates of *P. graminis*. Its resistance was, therefore, highly race-specific. Another variety, Kota, also showed good resistance to stem rust for a limited period but it soon succumbed to new races of the pathogen. Ceres, from the cross Marquis $\times$ Kota, became widely grown in North Dakota following its introduction in 1926, but in 1935 this variety became heavily infected with race 56 of *P. graminis* and millions of hectares of this variety were devastated by stem rust. During this severe epidemic, Thatcher, a new variety from Minnesota, was outstandingly resistant. Thatcher, which carries the stem rust resistance gene *Sr₅*, had been bred from the double-cross (Marquis $\times$ Iumillo durum) $\times$ (Marquis $\times$ Kanred). Each parent contributed genes for resistance to different individual physiologic races and, in addition, Iumillo probably contributed genes for adult-plant resistance (Stakman and Christensen, 1960). Although Thatcher is very susceptible to leaf rust, which is caused by *Puccinia recondita* (see page 99), it became widely grown and withstood well the effects of the stem rust epidemics of 1937 and subsequent years.

Recent work by Green and Dyck (1975) has indicated that the genetics of resistance in Thatcher is more complex than was previously thought. In experiments with a number of *P. graminis* races, Thatcher was susceptible to six races, moderately susceptible to seven other races and was resistant to two older races, both as a seedling and when adult. However, its resistance to these older races was not attributable to any of its known resistance genes.

Thatcher has been used as a parent in many breeding programmes. For example, it was crossed with Hope, a variety that had been developed many years earlier in South Dakota from a cross between Marquis and Yaroslav emmer (*Triticum dicoccum*), to produce a new variety named Newthatch. Hope, and a sister line called H-44, both of which carry the resistance gene *Sr*₁, are very resistant and have been extensively used as parents in breeding hard red spring wheats. Newthatch therefore inherited resistance genes from five sources: Marquis, Kanred, Iumillo durum, Yaroslav emmer and Hope. Such a combination of resistance genes in Newthatch and in other varieties derived from Hope and H-44 were expected to give a lasting control of stem rust. These varieties, together with two resistant durum varieties, Stewart and Carleton, did give excellent control of stem rust in the USA and Canada from 1939 until 1950, when a particularly aggressive physiologic race, 15B, first identified in Iowa in 1939, became widespread. The severe stem rust epidemics of 1950, 1953 and 1954 devastated varieties which had previously shown good resistance.

A stem rust-resistant spring variety, Selkirk, was developed in Canada during the early 1950s from a cross between Redman, a derivative of H-44, and the variety McMurachy, which carries the *Sr*₆ resistance gene (Peterson, 1958). Although Selkirk is susceptible to some races of *P. graminis* it has given a good control of stem rust in the field for many years. In 1963, when it occupied about 90 per cent of the hard red spring wheat acreage in the USA and Canada, isolates of race 32A, which is virulent on Selkirk, were identified at several sites. This variety was also completely susceptible to stem rust isolates in field plots in Mexico (Kernkamp, 1966). In spite of the excellent control previously given by this variety, it was assumed by many that, as the resistance of Selkirk is race-specific, its subsequent control of stem rust would be very limited. In practice, however, Selkirk has continued to show a high level of resistance in the field in Canada and the USA, although in glasshouse tests with seedlings Selkirk is moderately susceptible to several races of *P. graminis*. The susceptibility of Selkirk to leaf rust (*P. recondita*), however, caused this variety to be superseded in Canada during the late 1960s by other varieties, particularly Manitou and Neepawa. Although stem rust was common on susceptible varieties in Canada in 1971, no susceptible-type pustules of *P. graminis* (see Figure 3.14, page 77) were observed in fields of Manitou or Neepawa (Anonymous, 1972). Some of the most common isolates of the pathogen in 1971, however, included races C35 and C41, to which Manitou and Neepawa are susceptible as seedlings. Such races may be a threat to the rust resistance of these varieties, but no 'breakdown' in resistance has yet occurred.

Breeding for resistance to stem rust has also been important in Australia, where many different variants of *P. graminis* have been identified. Before 1940 the only variants in the field were those which had been present before the start of any wheat breeding programmes for their control. After 1940, however, the frequencies of certain genes for virulence in *P. graminis* were influenced by the frequencies of corresponding host genes for resistance in new varieties,

including Sr_6 , Sr_{11} , Sr_9b , Sr_9C and Sr_{17} (Watson and Luig, 1963). The first stem rust-resistant variety to be widely grown in Australia was Eureka, which carries the gene Sr_6 . When Eureka was released in 1938 it was resistant to all known races of *P. graminis* in Australia, but a new variant was identified in 1942 to which Eureka was susceptible, and the popularity of this variety declined in the mid-1940s. As the area sown with Eureka declined, the frequency of genes for virulence on plants with the Sr_6 gene correspondingly decreased, to such an extent that they were not detected in race surveys in 1959 and 1960. As a result of the disappearance of races to which it was susceptible, Eureka again became widely grown until a series of variants of *P. graminis* with genes for virulence on Sr_6 plants developed in the mid-1960s; these races eventually caused the withdrawal of Eureka from cultivation.

In 1941, when Eureka first became susceptible to stem rust, new varieties with other resistance genes were released in Australia. These included Gabo, Chater and Kendee, all of which carry the gene Sr_{11} . The corresponding gene for virulence in *P. graminis* increased in areas where these varieties were grown, and varieties with the Sr_{11} gene have now largely been replaced in Queensland and New South Wales by varieties with the Sr_9b , Sr_{17} and Sr_9C resistance genes. Watson and Luig (1963) concluded from the experience with the Sr_6 and Sr_{11} genes in Australia that, where cultivars have a simple genetic control of resistance, as in Eureka, a temporary withdrawal of those varieties from cultivation is of little permanent value in controlling stem rust. They also found that certain widely distributed variants of *P. graminis* had more virulence genes than were necessary for their survival, indicating that 'unnecessary' genes for virulence are not always lost. This conflicts with the views of Van Der Plank (1968), who suggested that when selection pressure is relaxed the frequency of unnecessary genes for virulence in pathogen populations declines.

Several wheats of African origin (including three varieties from Kenya and two from South Africa) have been sources of major genes for resistance to stem rust. The inheritance of resistance in these varieties, and in Veadeiro (a Portuguese variety), was studied by Knott (1957) in Saskatchewan. They each carry two or more of the resistance genes Sr_6 , Sr_7 , Sr_8 , Sr_9 , Sr_{10} , Sr_{11} and Sr_{12} . Veadeiro also probably carries two other genes for adult-plant resistance to race 15B, and modifying genes are probably present also in many of the African varieties. Most of these major genes have been exploited in many breeding programmes.

Although stem rust is usually much less severe in Europe than in North America and Australia, genes for resistance to *P. graminis* are common in European wheat cultivars (Bartoš, 1975). For example, Bezostaya I, which has been extensively grown in the USSR and Eastern Europe for many years, carries the Sr_5 resistance gene together with several other genes that condition adult-plant resistance to stem rust. Major resistance genes are widely distributed in modern varieties from most parts of the world, although their presence in many of these varieties has only recently been detected (McVey and Roelfs, 1975). For example, two new genes (Sr_{24} and Sr_{25}), which were apparently derived from *Agropyron elongatum*, have been found in the varieties Agent and Agatha (McIntosh, Dyck and Green, 1977).

Although resistant varieties have often given a worth-while control of the disease, such control has usually been ephemeral. This has been attributable almost entirely to 'breakdowns' in resistance following the widespread distribution of variants of *P. graminis* with genes for virulence corresponding to

resistance genes in the resistant cultivars. More than 300 distinct races of *P. graminis* have been identified by differential reactions on a few wheat varieties. This emphasizes the extreme variability of *P. graminis tritici*, variants of which can arise sexually on *Berberis* spp. (the alternate host) or asexually on graminaceous hosts, either by mutation or by somatic genetic recombination following exchange of nuclei. The relative importance of each source of variability is uncertain but experience in Australia, where *Berberis* is rare, suggests that the sexual stage of *P. graminis* is not very important in the production of new variants. On the other hand, resistance genes *Sr*₆ and *Sr*₁₁ have given a satisfactory control of stem rust in European countries where *Berberis* is uncommon but not in those countries where *Berberis* is prevalent (Špehar, Vlahović and Korić, 1976).

The very great genetic plasticity of *P. graminis* has meant that wheat varieties have been exposed to an almost infinite number of rust races. It is not surprising, therefore, that the control of stem rust by individual resistant varieties has usually been ephemeral; what is perhaps more surprising is that the resistance of some varieties has remained effective for so long.

Future possibilities

Knott (1971) has suggested that a permanent control of stem rust in North America may be possible by managing the evolution of the pathogen through appropriate use of varieties with general or race-specific resistance. He has advocated that the resistance gene *Sr*₆ should be used only in spring wheat varieties, and that general resistance should be used in winter wheats to avoid or reduce selection pressure on the pathogen. However, it would be difficult to obtain the co-operation of plant breeders in carrying out such a policy, which would be impossible to enforce. In any breeding programme which uses the *Sr*₆ gene it will be important to ensure that varieties also carry suitable minor genes for resistance. Experience in Australia has shown that resistance that is conditioned by *Sr*₆ is soon likely to be rendered ineffective by new resistance-breaking races of *P. graminis* if it is carried in susceptible genetic backgrounds, as in Eureka (Watson and Luig, 1963).

The use of multiline varieties to control stem rust has also been advocated (Borlaug, 1959; Knott, 1971). Such varieties would consist of a mixture of true-breeding lines, each having different genes for resistance to stem rust. It is claimed that, even if the resistance concerned was race-specific, selection pressure on the pathogen would not be strong because most races would be able to attack one or more lines and would therefore be able to survive. No single race would be able to attack more than a small proportion of the lines; this should ensure that no race would increase rapidly and cause severe damage. There are serious drawbacks to the concept of multiline varieties, however, particularly in view of the exacting rules, that exist in many countries, for genetic purity and uniformity of wheat varieties. Of even greater importance is the fact that the long-term advantages of multiline varieties in controlling stem rust have not been demonstrated convincingly on a large scale.

The recycling of major resistance genes has been suggested as a method of extending the period of usefulness of these genes in agriculture. Resistance genes, which have been matched by physiologic races of the pathogen, would

not then be used in the development of new varieties for a period of several years. After such a period had elapsed the virulence genes in the pathogen population might have disappeared, so that the resistance genes would again give an effective control. Experience with Eureka, however, suggests that recycling of resistance genes is of little value in controlling stem rust.

It is unlikely that major resistance genes derived from other *Triticum* species or related genera would provide a long-term solution to the stem rust problem. Luig and Watson (1976) transferred the *Sr*₂₇ resistance gene from rye (Imperial translocation line WRT 238.5) to susceptible bread wheats and found that the progenies were susceptible to some *P. graminis* populations: these populations were thought to be intervarietal hybrids between *P. graminis* f.sp. *tritici* and *P. graminis* f.sp. *secalis*. Such hybrids between *formae speciales*, if they occur in nature, might constitute a threat to wheat varieties with resistance genes from *Secale* and other alien sources, such as *Agropyron* (Knott, Dvorak and Nanada, 1977). Resistance derived from interspecific or intergeneric crosses will, therefore, probably be no more durable than intraspecific sources of major-gene resistance.

Origin of physiologic races

The results of genetic studies of host-pathogen relationships in stem rust generally conform with the gene-for-gene hypothesis developed by Flor (1956) and extended by Person (1959), which suggests that particular resistance genes in the host are matched by corresponding virulence genes in the pathogen. Loegering and Powers (1962) crossed two races of *P. graminis* and studied the pathogenicity of the F₁ and F₂ progenies on a set of differential wheat varieties. They identified eight independent virulence genes which they designated *P*₁–*P*₈, which corresponded to the resistance genes in the differential varieties. Avirulence in *P. graminis* was dominant in seven of the eight genes for pathogenicity and semidominant in *P*₆. Many more virulence genes in the pathogen have since been identified.

Virulence and avirulence of a pathogen such as *P. graminis* is controlled by specific genes whose products interact with corresponding resistance genes in the host according to a relatively simple genetic system (Watson, 1970). A mutation at a single locus in the pathogen will, therefore, often condition virulence to new host genotypes and this could result in a 'breakdown' of resistance in a variety. Random changes in the virulence of *P. graminis* to specific single host genes have occurred very frequently in North America, probably by mutation. Such changes permit the pathogen to attack varieties with resistance controlled by single genes but are not very effective in overcoming complex resistance that is controlled by several genes (Green, 1975). Many races of *P. graminis* with specific virulence to particular resistance genes and host genotypes have been identified in the USA (e.g. McVey and Roelfs, 1975; Roelfs *et al.*, 1977), in Canada (e.g. Green, 1975) and in Australia (e.g. Watson, 1970). Particular races are more common than others in different years and in different geographical areas; this depends partly on the differing distribution of varieties with specific resistance genes and partly on the degree of stabilizing selection which can occur in the field between different races of *P. graminis* (Osoro and Green, 1976). Some races of *P. graminis* seem to be less fit than others to survive or to compete (Ashour *et al.*, 1973).

Nature of resistance

Less is known about the nature of resistance to *P. graminis* than about its inheritance. The most widely used types of resistance seem to involve a hypersensitive reaction between host and pathogen (Knott and Anderson, 1956; Hooker, 1967). This is characterized by injury or rapid death of infected host cells and the inhibition of growth of *P. graminis*, although the pathogen is not necessarily killed (Santoso, Cheung and Willetts, 1973). Hypersensitivity may be a symptom of general stress in the host cells, caused by the presence of an incompatible genotype of a pathogen, rather than a cause of resistance (Mayama *et al.*, 1975). The hypersensitive responses of wheat to cereal rusts have been classified into different infection types or reaction types observed on the leaves after inoculation (Stakman and Christensen, 1960). These responses are usually classed according to six infection types depending on the number and size of rust pustules, and the amount of chlorosis and necrosis associated with them: these categories are; immune (no reaction); types 0, 1 and 2 (which indicate a high degree of resistance); and types 3 and 4 (which indicate susceptibility). In addition, there is an intermediate reaction type, designated X, where large and small pustules are interspersed on the same leaves (*Figure 3.14*, page 77). These reaction types have been used to distinguish between different levels of resistance to rusts that are expressed by different host genotypes, and also for identification of races of the pathogens by the reactions which they cause on sets of differential wheat varieties.

Caldwell (1969) has suggested that varieties such as Thatcher and Selkirk, whose resistance to stem rust gave an effective control of the disease for many years, may have expressed a combination of race-specific, hypersensitive resistance and 'general' resistance. For example, Thatcher possesses forms of mature-plant resistance derived from one of its parents, Jumillo, in addition to the major gene *Sr₅* which controls hypersensitivity. Selkirk has a form of general resistance to stem rust, derived from its H-44 ancestor through one of its parents, Redman, together with race-specific resistance, which is controlled by gene *Sr₆* from McMurachy.

The nature of such general resistance to stem rust is not understood. Some types of general resistance are expressed both in seedlings and adult plants. Goulden, Newton and Brown (1930) classified wheat varieties into three groups according to their resistance to stem rust as seedlings and mature plants: Group 1 consisted of varieties showing a similar disease reaction in seedlings and mature plants (e.g. Garnet, Marquis, Khapli and Jumillo); Group 2 comprised varieties in which there was a poor agreement between the resistance expressed by seedlings and mature plants (e.g. Hope and H-44); and Group 3 contained varieties in which there was incomplete agreement between seedling and adult-plant reactions (e.g. Reward, Kota and Marquillo). Adult-plant resistance in all three groups is apparently controlled by few genes and is inherited independently of seedling resistance. Guthrie (1966) found, in studies involving 70 wheat varieties, that seedling tests are not a reliable guide to the resistance of adult plants in the field. He concluded that field tests with adult plants in special nurseries were necessary to obtain reliable assessments of resistance to stem rust. Resistance that is expressed by adult plants but not seedlings sometimes involves hypersensitivity, and is not necessarily non-race-specific. 'Slow rusting', which is manifested as an increased generation time of *P. graminis* and by

the production of fewer pustules, apparently results from limitation of growth of the pathogen in the host, after penetration has occurred. This limitation is associated with reduced hyphal development in the host, reduced numbers of haustoria and more necrosis of host tissues (Martin, Littlefield and Miller, 1977).

Types of resistance to stem rust have been reported which apparently do not involve hypersensitivity; these include resistance to infection, resistance to spread of the pathogen in the plant and resistance to sporulation. Some varieties are more difficult to infect than others under uniform conditions of inoculation (Hayden, 1956). When plants of the varieties Sentry and Marquis were inoculated with light showers of *P. graminis* uredospores, an average of 0.02 stem rust lesions per culm developed on Sentry compared with an average of 5.25 lesions per culm on Marquis; with heavy spore showers the corresponding figures were 1.5 and 23.5 lesions per culm. Such differences in susceptibility to stem rust may be an important factor in the field resistance of certain varieties. Brown and Shipton (1964) also found very large varietal differences between the number of successful penetrations of stomata by *P. graminis* following uniform inoculation. Similar differences in 'infectibility' were also observed by Chakravarti and Hart (1959). The reasons for such differences are not understood but the stomata of some varieties open very slowly in the morning: this may decrease the number of successful penetrations by *P. graminis* in these varieties because the dew on the leaves, which is necessary for spore germination, will have dried by the time the stomata open. This hypothesis has been challenged, mainly on the grounds that *P. graminis* can penetrate closed stomata. Certain morphological characteristics have also been associated with resistance to stem rust. For example, rust fungi thrive best in thin-walled parenchyma tissue and certain cereal varieties develop thick-walled sclerenchyma tissue, especially in the culm, as they mature, and this may limit the spread of the fungus (Hart, 1931; Geshele and Babyanz, 1970). An unusually thick epidermis may also impede the development of rust pustules, thereby stopping or delaying sporulation. It is easy to select for such morphological characteristics in wheat (Skovmand, Wilcoxon and Heiner, 1977).

The growth of *P. graminis* in the leaves of some resistant varieties may be inhibited by antibiotic or toxic compounds produced in the host tissues. Ezekial (1930) found that sap from healthy resistant wheat varieties inhibited the growth of *P. graminis* germ tubes in hanging-drop cultures more than did extracts from susceptible varieties. The extracts from resistant plants retained their activity even when diluted 10000 times and after storage in a refrigerator for eight months. Ultrafiltration reduced the activity of the extract but paper filtration did not. This work was published in 1930 and, although the results have some important implications, no attempt appears to have been made to confirm these findings.

The balance of free amino acids in wheat seems also to be important in resistance to stem rust. Samborski and Forsyth (1960) floated inoculated, detached leaves of susceptible wheat varieties on solutions containing different purines, pyrimidines and amino acids, and found that histidine, isoleucine and methionine each inhibited the development of *P. graminis*; this inhibition could be reversed by glycine. Serine also inhibited the fungus, but glycine could not reverse this effect. Certain carbohydrates, including xylose, sorbose and sugar alcohols, also inhibited the development of stem rust. Pozsár and Király (1966) showed that the effect of rust infection resembled that of cytokinins because both cause an accumulation of glycine, increase protein synthesis

in wheat, and advance senescence. There may, therefore, be some connection between the accumulation of glycine near rust-infected areas and the apparent protective activity of glycine against the action of inhibitory free amino acids.

Prabhu and Swaminathan (1968) reported that wheat varieties which are resistant to *Puccinia* spp. are unusually susceptible to *Alternaria triticina*; the converse is also true. Resistance to *A. triticina* is associated with a high concentration of sucrose in the leaves and stems, and resistance to stem rust is therefore presumably associated with a low sugar content. This contention is supported by the effects on disease resistance of applications of 2,4-D and nitrogenous fertilizers, which are known to alter the sugar content of the leaves and stems.

Johnson *et al.* (1968) showed that a high protein content of the grain in wheat, an incompletely dominant character, is associated in adult plants with susceptibility to stem rust and resistance to leaf rust (*Puccinia recondita*). Adult-plant resistance to stem rust is therefore correlated with different levels of free amino acids, free sugars and grain protein but there is no evidence of a causal relationship between these factors and resistance.

Although so little is known about the nature of 'general' or 'generalized' resistance to stem rust, many varieties and potential varieties apparently show general resistance. For example, in Mexico, Sonora 64 and Siete Cerros were reported to be resistant to all known races at all growth stages in 1972. Particular attention has been given to the character known as 'slow rusting' which is seen as retarded development of disease. Wilcoxon, Skovmand and Atif (1975) grew several varieties in hill plots in the presence of single and mixed races of *P. graminis* and found that stem rust developed more slowly on Thatcher, McMurachy, Redman, Kenya 58 and Frontana than on the other varieties tested, including Baart, Prelude and Marquis. Similar varietal differences were obtained using inoculated detached leaves on benzimidazole. There was no relationship between slow rusting and the presence of the *Sr₅*, *Sr₆*, *Sr_{7b}* or *Sr₁₁* genes for specific resistance (Wilcoxon, 1976). Knott and Brennan (1976) crossed eight wheat varieties, which had shown durable resistance to stem rust, to produce four hybrids and found that each hybrid was resistant to all the *P. graminis* races against which they were tested; the inheritance of resistance was simple in one hybrid and complex in the other three. These results suggest that there are many independent factors which can contribute to slow rusting.

It is impossible to predict how durable such 'general' resistance would be if resistant varieties were grown on a large scale for several years. Nevertheless, it is reasonable to assume that varieties derived from others with durable resistance will themselves have a measure of durable resistance. The most effective and durable types of resistance to stem rust will probably be those that combine certain major resistance genes with minor genes which condition a high degree of general, adult-plant resistance. Recent work in North America has shown that even the most complex forms of resistance to stem rust can be used successfully in breeding programmes.

LEAF (BROWN) RUST

Leaf rust (*Puccinia recondita*), which occurs wherever wheat is grown, is the most widely distributed of all cereal rusts but is particularly important in parts of North America, Eastern Europe, the USSR, India, China and Japan. Leaf rust

can seriously reduce grain yields in susceptible varieties, yield losses of more than 35 per cent having been reported following early infection. As the name 'leaf rust' implies, the reddish-brown spore-producing pustules (uredinia) occur mainly on the leaves: they are scattered at random on both surfaces of infected leaves and consist of powdery masses of spores extruded through slit-like ruptures in the epidermis. The pustules, which are very variable in size or shape, are often surrounded by 'green islands', which are zones in which there is a higher concentration of chlorophyll than in other parts of the leaves. Severely diseased leaves become chlorotic and die prematurely, and serious attacks of leaf rust can lead to almost complete defoliation of susceptible varieties.

Breeding for resistance to *Puccinia recondita* has been an important objective in many parts of the world, and resistant varieties have played a major part in the control of the disease. Physiologic specialization in the pathogen, however, has enabled it to attack many previously resistant varieties. As with *P. graminis* and *P. striiformis*, races of *P. recondita* have been a major threat to the stability of resistance to leaf rust in many wheat varieties. In a monograph on wheat leaf rust, Chester (1946) pointed out that the variety Turkey, which had been listed in the late 1890s as being highly resistant to leaf rust, was one of the most heavily rusted commercial varieties in Oklahoma and Kansas in 1938. This apparent change in the susceptibility of Turkey to *P. recondita* was attributable to the increasing occurrence of variants of the pathogen to which Turkey is susceptible.

One of the earliest leaf rust-resistant varieties was Kanred, which became widely grown in parts of the USA after its introduction in 1917. Another variety, Kawvale, with moderate leaf rust resistance was released in Kansas in 1932. These two varieties were subsequently used as sources of resistance to *P. recondita* in many parts of the USA. Resistance from Warden, an old Australian variety, was introduced into soft red winter wheats.

The variety Hope and a sister line H-44, which were derived from a cross between *Triticum dicoccum* (Yaroslav emmer) and Marquis, have already been mentioned in connection with resistance to stem rust (see page 92). They showed a high level of resistance to leaf rust and were used very extensively as parents in breeding for resistance. For example, Hope was crossed with Thatcher, which is very susceptible to leaf rust, to produce Newthatch, which combined resistance to stem and leaf rusts. Shortly after its release, however, Newthatch was observed to be very susceptible to leaf rust, presumably because of the widespread occurrence of resistance-breaking races of *P. recondita*.

Several species of *Triticum*, including *T. timopheevi* and *T. durum*, are very resistant to leaf rust. Although *T. timopheevi* is a tetraploid, it has been successfully crossed with hexaploid wheats (*T. aestivum*), and several types of bread wheat incorporating resistance to leaf and stem rust derived from this species have been developed. Leaf rust-resistant varieties have been developed in many countries other than the USA, including Europe, the USSR, Australia and Canada; sources of resistance from the USA breeding programmes have commonly been used. The varieties Bezostaya I, Kavkas and Aurora, which have been grown over a large area of the USSR and Eastern Europe, had high levels of leaf rust resistance when they were first introduced. In the early 1970s, however, several variants of race 77, which are highly virulent on Kavkas and Aurora, were identified in the USSR (Voronkova, 1975) and in 1975 these variants predominated in the *P. recondita* populations on these varieties (Lesovoj,

Shkodenko and Pantaleev, 1976). In Canada, two of the most popular varieties during the 1950s were Lee and Selkirk which, at the time of their release, possessed good resistance to leaf rust: by 1960, however, Lee was susceptible and Selkirk was only moderately resistant (Anderson, 1961). Manitou, which largely replaced Selkirk in Canada, is more resistant to leaf rust, having adult-plant resistance derived from Frontana.

Caldwell (1969) cites other examples of gradual loss of resistance to leaf rust in wheat varieties derived from Chinese Spring (CI 6223) which have been widely grown in parts of the USA. These varieties exhibited a polygenically controlled, hypersensitive type of adult-plant resistance, but rust populations of increasing virulence finally overcame this resistance during the period 1961–1965.

Several genes that control resistance to leaf rust have been identified. These genes, designated Lr_1 , Lr_2 and Lr_3 by Ausemus *et al.* (1946) were found in the varieties Malakof, Webster and Fulcaster in 1926, and five others (Lr_4 – Lr_8) were later identified by Fitzgerald, Caldwell and Nelson (1957). Selkirk and Exchange have two resistance genes, Lr_{16} and Lr_{10} , for resistance to leaf rust (Choudhuri, 1958; Anderson, 1961), the varieties Lee, Gabo, Timstein, Mayo 52 and Mayo 54 apparently carrying Lr_{10} , but not Lr_{16} . In addition, Lee and Timstein have recessive genes at other loci which condition adult-plant resistance and Gabo has gene Lr_{23} (McIntosh and Dyck, 1975). Dyck, Samborski and Anderson (1966) showed that partially dominant genes Lr_{12} and Lr_{13} condition adult-plant resistance in Exchange and Frontana, respectively; both genes control resistance to a large number of races, but the presence of one or more modifying genes is necessary for the maximum expression of resistance. Several other genes conditioning seedling resistance to leaf rust have been reported (e.g. Anderson, 1966; Dyck and Samborski, 1969; Dyck and Kerber, 1977). The genetic control of resistance to leaf rust of Hope was investigated at Cambridge by Law and Johnson (1967) using intervarietal chromosome substitutions of Hope into the susceptible variety Chinese Spring. Of the 21 possible substitution lines, only the one carrying the chromosome 7B from Hope was resistant to leaf rust. Two loci (lr and lrm) were involved, lr being located on the long arm and lrm on the short arm of chromosome 7B; lrm behaved as a modifier to lr . Modifying genes on chromosomes 7A and 7D were also identified. Resistance to race 5 of *P. recondita* is controlled by an incompletely dominant gene in *Aegilops speltoides* and this gene has been successfully transferred to bread wheat (Dvorak, 1977).

Many physiologic races of *P. recondita* have been identified by their reaction on sets of differential wheat varieties with different known major resistance genes. In identification tests, reactions of seedlings of differential varieties to variants of *P. recondita* are recorded approximately two weeks after inoculation and usually are classified using an internationally agreed system (Johnston and Browder, 1966). The most commonly used categories are listed below:

- o; minute chlorotic flecks
- O; large chlorotic flecks, no sporulation
- 1—; minute sporulating pustules surrounded by well-defined chlorotic areas
- 1c; small sporulating pustules surrounded by large chlorotic areas

- X+, X, X—; mesotheric reactions with pustules of varying sizes on the same leaf; + indicates a predominance of large pustules; — indicates a predominance of small pustules
- 3+; susceptible reaction, pustules not quite full size
- 4; fully susceptible reaction, large round pustules, no chlorosis.

In the USA, three sets of differential varieties have commonly been used to identify races: a Test Variety Set (consisting of about 20 varieties), a Universally Resistant Set (6 varieties) and a Supplementary Set (5 varieties) (Young and Browder, 1965). Over 100 apparently distinct physiologic races of *P. recondita* have been identified using these differential varieties. In more recent studies, sets of near-isogenic lines, each carrying a different resistance gene, have been used to identify virulence genes of *P. recondita* (Bošković and Browder, 1976). For example, samples of *P. recondita* populations from 24 European and Mediterranean countries, from 32 States in the USA and from Canada were tested against nine near-isogenic wheat lines with eight resistance genes. More than 60 per cent of the samples from Europe were virulent on all these lines, whereas samples from the USA and Canada were virulent on only two lines. This suggests that the genetic variability of *P. recondita* is much greater in Europe than in North America, although the reasons for this are not clear. The results of other international pathogenicity surveys of *P. recondita* using populations of the pathogen collected from many parts of the world have been summarized by Bošković (1976).

As has already been mentioned, this genetic variability in *P. recondita* has meant that many varieties, such as Turkey, Newthatch, Lee and Selkirk, which showed good resistance when released, subsequently succumbed to virulent races of the pathogen (Samborski and Dyck, 1976). In the case of Selkirk, however, a resistance gene still conditions moderate resistance to a wide range of races of *P. recondita*, and this protects the variety from severe attacks of leaf rust; it is uncertain, however, how long such resistance, or the adult-plant resistance in varieties such as Lee, Timstein and Manitou, will remain effective. Some types of adult-plant resistance to *P. recondita* are apparently non-race-specific, whereas others are effective only against certain physiologic races; it has not been possible to predict which types are expressed by particular varieties.

Some combinations of major resistance genes are still effective in controlling leaf rust in many parts of the world, in spite of the great genetic variability of *P. recondita*. In India, varieties carrying combinations of *Lr*₁, *Lr*₁₀, *Lr*₁₃ and *Lr*₁₄ showed good resistance to leaf rust when others carrying only one of these genes were badly attacked (Rao, Pahuja and Balakotiah, 1972). Over a four-year period the gene *Lr*₉ conditioned a high level of resistance even when other resistance genes were absent; *Lr*₉ and *Lr*₁₉ are the only effective genes for resistance to leaf rust in the USSR today (Krivchenko, 1977).

The cytoplasm can affect the expression of major genes for resistance to *P. recondita* and this may account for differences in the effectiveness and durability of individual resistance genes in different genetic backgrounds. Washington and Maan (1974) found that genes of the variety Chris, which normally confer resistance to leaf rust at the adult-plant stage, did not do so with some races of *P. recondita* in the presence of cytoplasm from one of several alien species, including *Triticum timopheevi* and *Aegilops speltoides*. This suggests that certain

alien cytoplasm can alter the expression of major genes for resistance to leaf rust.

Very little is known about the nature of resistance to leaf rust, although a distinction has often been drawn between 'seedling' and 'adult-plant' resistance. Much of the seedling resistance, particularly that which is conditioned by major genes, probably involves hypersensitivity, as do certain forms of adult-plant resistance (Caldwell, 1969). For example, the resistance of Thew, which is controlled by resistance gene *Lr₂₀*, is associated with physiological changes leading to a collapse of host protoplasts and a loss of cytoplasm in the hyphae of incompatible races of *P. recondita* (Jones and Deverall, 1977). These types of resistance have usually given only a temporary control when used in varieties that have become widely grown.

Types of resistance other than hypersensitivity have also been identified and some of these are probably less race-specific and more durable. Such 'general' resistance may involve a tendency to escape infection, caused either by a reduced rate of spore germination on the leaf surface or by the exclusion of the pathogen by the stomata (Romig and Caldwell, 1964) and also the inhibition of the growth, development and sporulation of the pathogen. Caldwell *et al.* (1957) found that the variety Dual (CI 13083) showed a strong tendency to escape leaf rust infection; for example, when 90 per cent of plants of a susceptible variety, Trumbull, became infected, Dual had only 4 per cent of plants infected. Zadoks (1972) analyzed components of resistance to *P. recondita* in a number of varieties and found that, under uniform conditions of inoculation, there were very large varietal differences in the number and size of pustules which subsequently developed. He also found that the ratio of penetrations to pustules per unit of leaf area varied with variety; for example, this ratio was 1.01 in Joss Cambier and 0.57 in Rubra. There were also varietal differences in latent period, sporulation rate, rate of lesion growth and infection period. Zadoks suggested that some of these components might be non-race-specific.

Tolerance to leaf rust has also been reported, similar amounts of leaf rust causing different losses of grain yield in different varieties. For example, some hybrid families with 65–100 per cent of rusted leaf area suffered 44 per cent loss of yield in field experiments, while other families with similar amounts of rust infection lost only about 10 per cent of their potential yield (Lukyanenko, 1934). Tolerance might give a more lasting control of the effects of leaf rust than resistance that is based on hypersensitivity, because 'tolerance-breaking' races of the pathogen would have no competitive advantage over established races (Caldwell *et al.*, 1958).

Several workers have suggested that some mechanisms of resistance to leaf rust involve specific changes in protein synthesis in the host plant. The role of specific proteins in resistance mechanisms is not known, but they may act as enzymes or antitoxins, or as specific nutrients for the pathogen (Shaw, 1963; Dunin and Budanov, 1972; Guseva and Gromova, 1976). Environmental factors that tend to increase the protein content of wheat also increase susceptibility to leaf rust, and Majstrenko (1967) observed a negative phenotypic correlation between high gluten content of the grain and good resistance to leaf rust; this may have been attributable in part to an association between high gluten content and late maturity and consequently long exposure to rust attack. On the other hand, Johnson, Schmidt and Mattern (1968) found that, in the variety Atlas 66, there is an incompletely dominant gene for high protein content which is

closely associated with leaf rust resistance. High protein selections from crosses between Atlas 66 and Comanche or Wichita were predominantly resistant to leaf rust at the adult stage.

Although there are so many conflicting views on the nature of 'general' resistance to leaf rust, considerable progress has been made in breeding wheat varieties with broadly based resistance. Such varieties are usually termed 'slow rusting' because leaf rust epidemics in the field develop much more slowly on them than on other varieties (Caldwell, Roberts and Eyal, 1970). General resistance to *P. recondita* has been reported in many countries including the USA (Caldwell *et al.*, 1970), India (Verma, Kumar and Sudhir Kochar, 1975), the USSR (Merezhko, Trubchaninov and Borodanenko, 1974) and Romania (Ionescu-Cojocaru and Negulescu, 1976). It remains to be seen whether the resistance of varieties which incorporate general resistance is truly durable when they are grown over a large area.

Barley

POWDERY MILDEW

Powdery mildew (*Erysiphe graminis* DC. f.sp. *hordei*) is probably the most important fungal disease of barley. It occurs wherever barley is grown and can attack plants at all stages of growth. The effects on grain yield can be serious and losses of nearly 50 per cent have been reported in late-sown crops of susceptible spring barley varieties. In susceptible barleys, powdery mildew causes chlorosis of the leaves because infection reduces the number of polysomes in the chloroplasts (Dyer and Scott, 1972). The growth of the roots is also significantly retarded because mitotic division at the root tips of infected plants is inhibited (Minarić and Paulech, 1975).

Although very effective systemic fungicides are now available to control powdery mildew, the use of resistant varieties is probably the main control method and is likely to remain so. Variants of *E. graminis* f.sp. *hordei* that are resistant or tolerant to certain fungicides have been identified, and such variants may limit the effectiveness of chemical control in the long term (Wolfe, 1971). Pesticide residues have also been found in the grain, leaves and stems of fungicide-treated plants (Waring and Wolfe, 1975) and too much reliance on chemical control of powdery mildew may have undesirable effects on the environment.

Biffen (1907) pioneered breeding for resistance to powdery mildew when he showed that the resistance of *Hordeum spontaneum*, a wild relative of barley, is controlled by a single recessive gene. Ten resistance genes (eight dominant and two recessive) had been identified by the mid-1950s (Schaller and Briggs, 1955); five of these genes were found to occur on chromosome 5, two at a single locus, designated *Ml-a*. Later work showed that there are also numerous genes for resistance to *E. graminis* at other loci. Moseman (1966) concluded that wild and cultivated barleys carry an almost unlimited number of genes that condition resistance to powdery mildew, and that many of these genes are at complex loci (e.g. *Ml-a*).

Sources of host resistance to powdery mildew of barley have been reviewed in detail by Wolfe (1972) and Wiberg (1974a; 1974b). Many of the most widely used sources were collected at different times, from about 1930 onwards, from

Asia (particularly Asia Minor), the Near East, Ethiopia and Somaliland. Sources of resistance from these collections have been used extensively in research and breeding programmes in Europe, North America and Japan.

The genetics of resistance to powdery mildew is one of the most thoroughly studied features of higher plants. Eleven alleles (designated *MI-a*₁ to *MI-a*₁₁) at or near the *MI-a* locus have been identified by Moseman and Jørgensen (1971; 1973) and there are probably further alleles at this locus (Wolfe, 1972). At least seven loci conditioning resistance have been recognized, but only some of the genes at these loci are being exploited in current breeding programmes. These include *MI-a*₇ (derived from Lyallpur 3645), *MI-a*₄ (from HOR 1063), *MI-a*₉ (from Monte Cristo), *MI-g* (from Goldfoil), *MI-h* (from Hanna) and *MI-p* (from Nigrate). The resistance of Maris Mink and Sultan depends partly on the gene *MI-a*₁₂ from Arabische. In addition, two resistance genes from Ragusa lines have been widely used, and genes conferring incomplete resistance derived from *Hordeum laevigatum* have been exploited in several European varieties including Vada, Minerva and Universe. The inheritance of resistance to powdery mildew is probably more complex than was previously believed, and it is now thought that minor genes often modify the expression of resistance conditioned by major genes.

Induced mutants of barley that are resistant to powdery mildew have been derived from several different parents (Jørgensen, 1971; Wolfe, 1972; Wiberg, 1973). Genetic studies have shown that this resistance is conditioned by a series of allelic recessive genes at the *ml-o* locus on chromosome 4. These mutant lines were at first thought not to occur spontaneously, but Wolfe and Finch (1973) have subsequently shown that resistance genes at the *ml-o* locus are present in several Ethiopian barleys. These alleles condition an unusual type of chlorotic and necrotic spotting on the leaves and are associated with low grain yield. Nevertheless, many plant breeders are trying to incorporate the *ml-o* gene into their breeding material because the mutant barley lines are resistant to all known races of *E. graminis* f.sp. *hordei*. Although lines carrying the *ml-o* gene have expressed an equally high level of resistance at 78 centres in 29 countries (Jørgensen, 1977), there is no guarantee that resistance conditioned by *ml-o* alleles will be any more durable than other types of major-gene resistance.

There are many commercial barley varieties which carry major genes for resistance to powdery mildew, either individually or in various combinations (Table 4.1). Most of these genes condition resistance which is highly race-specific, (Figure 4.5) and new varieties carrying them have generally selected their own pattern of *E. graminis* races (Wolfe, 1972). Before 1950, populations of *E. graminis* f.sp. *hordei* in Europe were probably not capable of attacking the present standard set of differential barley varieties. The situation changed, however, when barley varieties with the resistance gene *MI-g* became widely grown in the mid-1960s (Howard *et al.*, 1970). This gene occurs in many varieties including Union, Cambrinus, Impala, Deba, Abed, Zephyr, Mosane, Julia and Inis. Physiologic race surveys in the UK showed that most isolates of *E. graminis* f.sp. *hordei* which were collected in England during the period from 1964 to 1969 were able to attack varieties with the *MI-g* resistance gene, either alone or in combination with the *MI-a*₆ gene (Table 4.1). The popularity of these barley varieties declined in the UK as the frequency of *E. graminis* isolates which could attack them increased. A simple but effective technique of

Table 4.1 PERCENTAGE FREQUENCY OF RACE GROUPS OF *ERYSIPHE GRAMINIS* F.SP. *HORDEI* ON BARLEY IN THE UNITED KINGDOM, 1964–1969.*

Host genes attacked by <i>E. graminis</i> isolates with corresponding virulence genes	Barley cultivars attacked	Percentage attack					
		1964	1965	1966	1967	1968	1969
<i>Ml-g</i>	Zephyr, Julia, Deba Abed	38	32	39	5	7	8
<i>Ml-a₆</i>	Midas	10	9	5	0	1	0
<i>Ml-g + Ml-a₆</i>	Impala, Zephyr	0	5	28	75	84	65
<i>Mla'12</i>	Sultan, Emir, Hassan	0	0	0	0	3	19**

* Data from Howard *et al.*, 1970

** Widespread attacks of powdery mildew were reported on Sultan after 1969

conducting race surveys, involving the use of mobile nurseries, has recently been described by Wolfe and Minchin (1976). Such techniques will help to improve the efficiency and flexibility of surveys to identify virulence genes of *E. graminis* in the field. The physiologic race situation in continental Europe, North America and Japan has also been surveyed in detail and is essentially similar to that in England (Nover *et al.*, 1968; Moseman, 1968; Wiberg, 1970).

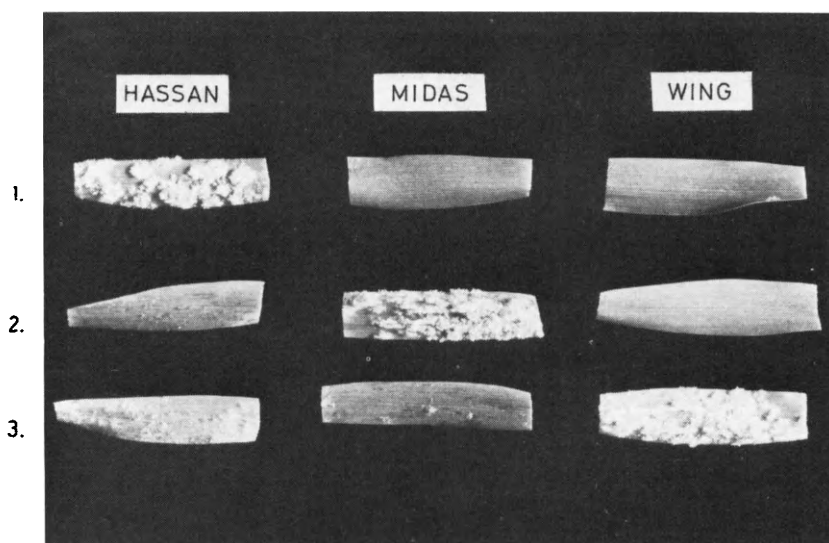


Figure 4.5 Race-specific reactions of three spring barley varieties (Hassan, Midas and Wing) to three isolates of *Erysiphe graminis* f.sp. *hordei*, the powdery mildew fungus. (By courtesy of Dr M.S. Wolfe, Plant Breeding Institute, Cambridge)

It is clear that *E. graminis* f.sp. *hordei* has a very large capacity for variation. The importance of the sexual stage in promoting this variability is uncertain, although cleistothecia (fruiting bodies) seem to be the principal means of 'over-summering' by the pathogen in hot, arid regions. Under experimental conditions, crosses between two races of *E. graminis* f.sp. *hordei* yielded ascospores of 14 races in addition to those of the parent races; four of these races had not previously been reported (Al-Ani, 1969). This shows that the sexual stage can be a very effective source of genetic variation, but sexually produced ascospores probably do not contribute greatly to the spread of powdery mildew in moist, temperate countries (Koltin and Kenneth, 1970). Most of the very considerable genetic variability of *E. graminis* f.sp. *hordei* must therefore be attributable to somatic mutations and asexual genetic recombination.

The result of this genetic variability of *E. graminis* has been to deplete very rapidly the number of resistance genes that give an effective control of powdery mildew. Nevertheless, there are still many resistance genes which have not been exploited so far, and new sources of resistance are still being found in cultivated barleys (e.g. Caddel, 1976), and in their wild relatives, for example *Hordeum bulbosum* (Jones and Pickering, 1978). It would be wasteful, however, to use them haphazardly in breeding programmes as has been done in the past, and new strategies are being explored. Whitehouse (1969) suggested that there should be an international agreement on the sequence of resistance genes to be released and a predetermined maximum permitted acreage of individual varieties with race-specific resistance. Such an agreement would be difficult to enforce, however, and it is necessary to find new ways of controlling powdery mildew by resistant varieties, including the use of general or 'partial' resistance (Slootmaker, 1972).

There is very little published information concerning non-race-specific resistance to powdery mildew in barley. Resistance remaining after major genes have been overcome has been noted in several varieties. For example, varieties which carry the *ML-g* resistance gene differ in degree of resistance to isolates of *E. graminis* f.sp. *hordei* which carry the corresponding gene for virulence. The varieties Zephyr and Mosane are very susceptible to such isolates and Union, Swallow and Deba Abed are moderately resistant (Howard *et al.*, 1970). Similarly, Maris Canon is more resistant in the field than Impala or Inis, although all three varieties have the same two major resistance genes, *ML-a₆* and *ML-g*. Another variety, Asse, which has shown good field resistance to powdery mildew, apparently carries no known major resistance genes (Frimmel, Schwarzbach and Fishbeek, 1975). Physiologic races virulent against Vada and its derivatives have been common in Europe for several years, but they do not seem yet to have caused severe epidemics on these varieties (Moore, 1977). Vada, which carries a major resistance gene from *Hordeum laevigatum*, may therefore have some form of non-race-specific resistance which continues to protect it and related varieties against bad attacks of powdery mildew.

The mechanisms of race-specific resistance to powdery mildew have been studied by workers in many parts of the world. Although it is generally agreed that a hypersensitive response of penetrated host cells is usually involved in incompatible reactions, the mechanisms are complex and imperfectly understood (Ellingboe, 1972). The development of *E. graminis* f.sp. *hordei* on the leaves of resistant barleys, and the events which lead up to the death both of infected host tissues and of the pathogen, have been studied in considerable detail by

many workers (e.g. Benada, 1970; Mount and Slesinski, 1971; Stanbridge, Gay and Wood, 1971; Bracker and Littlefield, 1973; McKeen and Rimmer, 1973). Hirata and Togashi (1957) demonstrated that, if the development of the first haustorium of *E. graminis* is checked, secondary haustoria are not produced and there is no subsequent development of the pathogen. They suggested that specific host-pathogen interactions, which impede the first haustorium, may involve proteins, probably enzymes, the synthesis of which is linked with the polyphenol oxidase system. Bushnell, Dueck and Rowell (1967) confirmed the importance of the first haustorium in powdery mildew and found that removal of the first haustorium stopped hyphal growth immediately. Although growth was sometimes resumed, it was very slow and new haustoria were not formed. Masri and Ellingboe (1966) compared the infection process on barleys with the resistance genes *Ml-a*, *Ml-g*, *Ml-k* and *Ml-p* with that on a susceptible variety, Manchuria. All these major genes had more than one of the following modes of action on the pathogen: (1) exclusion of the pathogen from the host cell; (2) delay in early haustorial development; (3) distortion or destruction of most haustoria within five days after inoculation; (4) suppression of sporulation. However, different resistance alleles at the same locus appear to act in the same way to produce a resistant reaction, although they can be overcome by different allelic or non-allelic virulence genes in the pathogen. These infection processes do not, therefore, explain the phenomenon of race specificity.

McKeen and Bhattacharya (1970) found that 73 per cent of conidia of an avirulent isolate of *E. graminis* f.sp. *hordei* which germinated on coleoptiles of barley carrying the *Ml-a* gene, produced penetration pegs which passed through the epidermal wall. Only a small proportion of these produced haustoria and secondary hyphae, however, and conidiophores were not formed. After about five days, growth ceased and the colony died. These results, and those of Ellingboe (1972) suggest that there are two main stages of major-gene resistance to powdery mildew, one effective during penetration and the other two to four days later. The earlier stage may coincide with a period of maximum phytoalexin activity which occurs about 12 hours after inoculation in incompatible reactions (Oku *et al.*, 1975). The fact that phytoalexins occur in tissues surrounding colonies of *E. graminis* on leaves of compatible hosts suggests that production of phytoalexins may be quicker and more efficient in resistant than in susceptible barley genotypes.

Many types of resistance to powdery mildew which do not seem to be associated with resistance conditioned by known major genes, have been reported. For example, some barley cultivars seem to be less damaged by the disease than others and such tolerance could be exploited in breeding programmes. Varietal differences in several resistance components, including resistance to spore deposition, inhibition of spore germination, inhibition of the growth, development and sporulation of the pathogen and an increased generation time, have been found between barley cultivars with the same major resistance genes (Russell, Andrews and Bishop, 1975, 1976; Russell and Muszanskyj, 1976). Benada (1970) found that in a susceptible variety, Valticky, the proportion of germinated conidia from which haustoria developed varied at different temperatures and with different nutrient treatments of the host. Neighbouring epidermal cells often showed different degrees of susceptibility, and in some cells granular bodies developed under abortive appressoria; these may have contributed to the

resistance of the cells. These findings suggest that some resistance mechanisms operate even in varieties that are classified as susceptible, and that their effectiveness may be strongly influenced by environment. Such mechanisms may help to explain the observation that specific resistance genes may still influence the survival of the pathogen even when they have been overcome by specific virulence genes.

Resistance to powdery mildew is associated with differences in redox potential (Benada, 1966) and with differing concentrations of sugars and other carbohydrates (Russell and Hudson, 1973) in the leaves. Edwards (1970) observed a protein-like substance which was apparently produced in the host epidermal cells in response to infections; this substance was confined to the papilla in susceptible cells, but diffused throughout the penetrated cell and to adjacent mesophyll cells in resistant plants. The significance of this substance in relation to resistance is not known. Yang and Ellingboe (1972) found that only malformed appressoria of *E. graminis* occurred on plants with mutations which affected the wax layer on the leaf surface. Other genetically controlled morphological characters such as the thickness of the cuticle and of the epidermal cells, have also been correlated with certain kinds of resistance to *E. graminis* in barley (Eizenberga, 1974; Horváth, 1974).

More information about types of resistance to powdery mildew that do not involve hypersensitivity is urgently needed. Although there is no certainty that any of these types will be non-race-specific, they will provide the breeder with much-needed additional means of increasing the durability of resistance in future varieties.

Future possibilities

The use of single, major genes for resistance to powdery mildew in barley varieties has not usually given a lasting control of the disease, and the use of major genes in known combinations has given similar disappointing results. *E. graminis* has been able to develop new variants with virulence genes corresponding to resistance genes in the host, thus leading to 'breakdown' of resistance in many varieties. Efforts to breed varieties with long-lasting resistance to powdery mildew, through the uncontrolled use of small numbers of major resistance genes, will probably be unsuccessful. This view is shared by many breeders and several new approaches to the problem are being investigated. One possibility is to regulate the sequence of the specific resistance genes used in varieties, in the hope that this will increase the durability of resistance. A second possibility is to use resistance genes in certain combinations which appear to select for less efficient forms of the pathogen (Wolfe, 1972; Wolfe and Barrett, 1977). For example, 'Maris Mink' (a spring barley) carries several major resistance genes and has continued to give a good control of powdery mildew for many years; development of most isolates of *E. graminis* is impaired on this variety and little sporulation of the fungus occurs as a result (Figure 4.6).

A third approach would be to recycle specific resistance genes by withdrawing from agriculture, for several years, those varieties which carry them. Although this idea is attractive in many ways, experience with the recycling of genes for

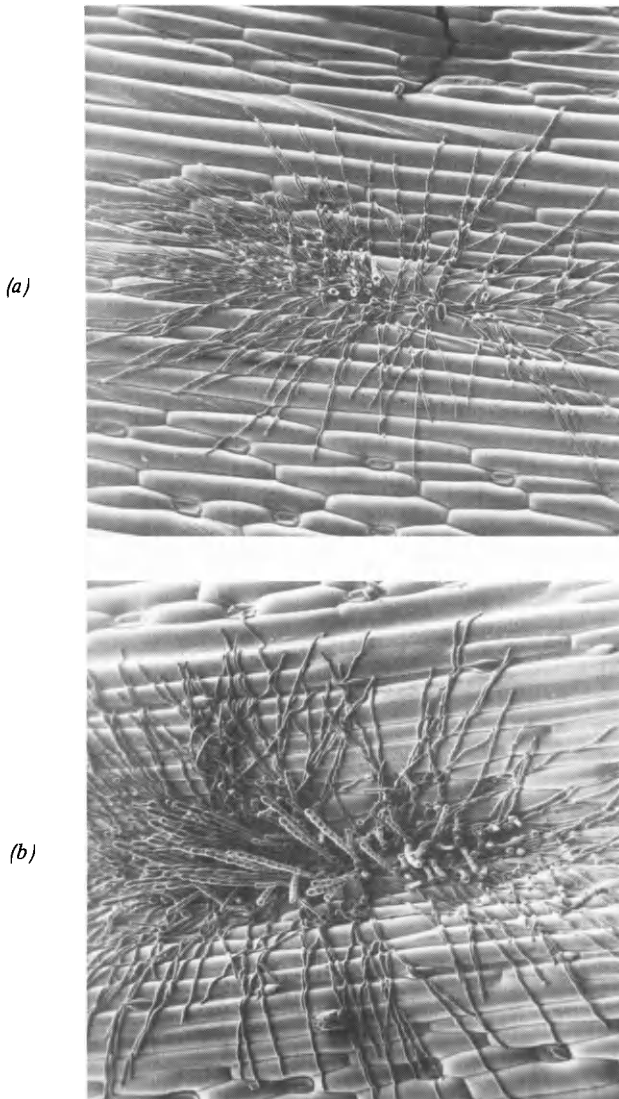


Figure 4.6 Scanning micrographs of the development of *Erysiphe graminis f.sp. hordei* on leaves of (a) a resistant variety (Maris Mink) and (b) a susceptible variety (Proctor). There is much less mycelium and sporulation on Maris Mink, which carries several resistance genes, than on Proctor which has no major resistance genes. (By courtesy of Rothamsted Experimental Station)

resistance to stem rust of wheat in Australia (see page 93) suggests that this would not be very effective. A fourth possibility is to use multiline varieties, as suggested by Borlaug (1959) and Frey, Browning and Simons (1977). Such varieties would consist of a number of near-isogenic lines, each carrying a different resistance gene; individual lines would be replaced when they became susceptible. Although multiline varieties have given a good control of powdery

mildew under some experimental conditions, there are many problems involved in producing and handling varieties which consist of genetically distinct components. Many of these problems do not apply to the use of mixed plantings of different varieties which each carry a different gene or combination of genes for resistance to *E. graminis*. Preliminary field experiments of such varietal mixtures in the UK have given encouraging results. Such variety mixtures are unlikely to result in the production of 'super races' of *E. graminis* which could attack all constituents of varietal mixtures.

A fifth possibility, and perhaps the most promising in the long term, is to search for types of resistance which are non-race-specific or which cause only a small response towards increased virulence in the pathogen. Although such types of resistance may give only a partial control of powdery mildew, their use may decrease mildew damage to an acceptable level and enable the breeder to concentrate on important objectives other than disease resistance. It may, eventually, be possible to combine race-specific hypersensitivity with types of partial resistance in the same variety, and thereby to achieve a high level of durable resistance to powdery mildew. In the meantime, combinations of resistance genes, either in single varieties, multiline varieties or varietal mixtures, together with applications of appropriate fungicides, should give an adequate control of the disease.

Rice

BLAST

Blast (*Piricularia oryzae*) is probably the most serious fungus disease of rice. It is common in most of the humid paddy regions of the world, but not in areas of low relative humidity, such as California, where rice is grown under irrigation (Ou, 1972). Small, bluish flecks which develop into brown spots with grey centres appear on leaves of diseased plants; these spots may enlarge and coalesce to such an extent that the leaves die. Lesions can also develop on the culm and near the base of the panicle. The yield of grain can be severely reduced by early infection in susceptible varieties.

The disease is seed-borne and can be spread by air-borne conidia. Control measures include dressing seed with fungicides, spraying the foliage with fungicides and the use of resistant varieties. Toriyama (1972) reports that, in Japan, the use of certain fungicides has created many problems, including direct toxicity to the farmers who apply the sprays or dusts, residual toxicity in food, and environmental pollution. For these reasons and for economy, great attention has been paid to the breeding of blast-resistant varieties. There is also the danger of the widespread occurrence of fungicide-tolerant forms of *P. oryzae* (Hwang and Chung, 1977).

In Japan, many crosses have been made between native varieties in attempts to breed resistant varieties. Iwatsuki (1942) crossed lowland rice varieties with Japanese upland varieties, which have a much higher level of resistance, and produced several highly resistant varieties, including Wakaba and Ukon-nishiki, for use in south-western Japan. The genetics of resistance in these varieties is not fully understood although they all carry the major resistance gene *Pi-a*.

Many other resistant varieties have since been produced, including Norin 22, Norin 23, Yamabiko, Rikuu 132, Fuginori and Ishikarishiroke (Toriyama, 1972). The resistance of some of these varieties is controlled by major resistance genes *Pi-i* and *Pi-a* and in others by polygenes. Although Yamabiko and Fuginori carry the resistance gene *Pi-a* they are susceptible to many widely distributed races of *P. oryzae* with the corresponding gene for virulence. Some Chinese varieties of the Japonica type, which carry resistance gene *Pi-k*, were very resistant to blast and were crossed with Japanese indica types to produce the varieties Kusabue, Yuukara and Senshuraku, which combined high yield good grain quality and, initially, good resistance to blast. Within three to five years after their release, however, these three varieties were very severely damaged by blast because they were attacked by races of *P. oryzae* with the corresponding virulence gene for *Pi-k*. Other varieties carry a resistance gene *Pi-m* (from a Chinese variety, Hotushitahmi) in addition to *Pi-k*; some of these also inherited field resistance from some of the parental Japanese native varieties. A few varieties, in which major-gene resistance and field resistance are combined, have shown a moderate level of resistance even to races that are virulent on varieties with the *Pi-k* gene (Ezuka *et al.*, 1969). Other resistance genes which have been used in breeding programmes in Japan include *Pi-ta* and *Pi-z*, the latter gene being derived from a US variety, Zenith. Races of *P. oryzae* with the virulence gene corresponding to *Pi-z* have already been identified, but *Pi-ta* seems to condition a more stable form of resistance (Toriyama, 1972).

Several other sources of resistance to blast, in addition to those used in Japan, have been identified at the International Rice Research Institute (IRRI) in the Philippines (Khush and Beachell, 1972; Ou, 1972). Some varieties, including Tatep, have shown an unusually broad spectrum of resistance to blast races; these varieties have been crossed with high-yielding dwarf selections (Ou *et al.*, 1971), and a few, including IR20, are resistant to blast, tungro virus, bacterial leaf streak and the green leafhopper (Pathak, Beachell and Andres, 1973). Although the resistance of Tatep is stable it is not 'horizontal' or 'non-race-specific' because this variety reacts differentially to different races of *P. oryzae* (Ou, 1974; 1977).

The stable resistance of varieties such as Tatep, including several from Japan (Yunoki *et al.*, 1970), is characterized by the development of few *P. oryzae* lesions on the leaves under conditions when many lesions develop on susceptible varieties. Japanese workers have distinguished between two categories of resistance to blast, namely 'true' resistance and 'field' resistance (e.g. Kiyosawa, 1972). 'True' resistance seems to be synonymous with vertical or race-specific resistance and usually involves a hypersensitive reaction to the pathogen, controlled by a single major gene. In testing for 'true' resistance, plants have been inoculated with appropriate isolates of *P. oryzae*, either by injecting or spraying them with suspensions of spores in water. Plants on which no lesions develop are considered to be resistant and are selected for future breeding. Unfortunately, the results of spraying and injection techniques of inoculation do not always agree; in addition, different results can be obtained from tests with seedlings and adult plants. For example, the variety St 1 was susceptible to seven standard races of *P. oryzae* when inoculated by injection, but only a few blast lesions were produced on this variety when it was inoculated with these races by the spray method. Other methods of screening for resistance to blast disease have been described by Ou (1971) and Marchetti (1975).

Selecting and testing for resistance to rice blast has often been carried out in naturally infected field experiments. Field trials in 26 countries over a period of nine years showed that many varieties possess field resistance that is effective against a broad spectrum of naturally occurring isolates of *P. oryzae* (Ou, 1974). Breeding lines with the fewest blast disease lesions were found to be resistant to more isolates of the pathogen than were other lines (Ou, Nuque and Bandong, 1975). Varietal differences in lesion number were reliable indicators of differences in race-specific 'true' resistance.

The relationship between 'true' resistance and field resistance to blast is uncertain. The situation is further confused by differences in the terminology used by different workers. For example, it is difficult from the literature to differentiate between the 'quantitative' resistance of Ou *et al.*, (1975), the 'field' resistance of Ezuka (1972), the 'stable' resistance of Ou (1974) and the 'horizontal' resistance of Sakai and Kono (1974). Ezuka (1972) reported that field resistance is usually non-race-specific, is more affected by environmental factors than 'true' resistance and is controlled polygenically in most varieties but oligogenically in others. Sakai and Kono (1974) reported that several different resistance mechanisms contribute to the 'horizontal' resistance of Norin 22 and Akitsicho, including resistance to penetration by *P. oryzae* and reduced sporulation of the pathogen.

Phytoalexins are apparently produced by rice in incompatible reactions with *P. oryzae* (Uehara, 1958). Differences in the chemical constitution of resistant and susceptible varieties have also been reported; low concentrations of nitrogen and high sugar content of the leaves are associated with susceptibility to rice blast in some varieties (Reddy, Kothandaraman and Mahadevan, 1969; Baek, 1970).

The very considerable genetic variability of *P. oryzae* has resulted in the rapid adaptation of the pathogen to many varieties with 'true' resistance to rice blast. Wu and Tsai (1974) have shown that new mutations of *P. oryzae* can readily be induced by ultra-violet irradiation. These workers suggest that the occurrence of recombinants among haploids segregating from diploids indicates that parasexuality also contributes to variation in the pathogen.

Future possibilities

In spite of the ability of *P. oryzae* to develop resistance-breaking races, Tatep, Carreon and several other rice varieties have consistently shown a high level of resistance to blast in several parts of Asia, Africa and Latin America. However, no rice variety is resistant to all known races of the pathogen, although some varieties are resistant to most (Ou, 1977). The 'field' resistance of Japanese varieties is, therefore, not horizontal or non-race-specific. Nevertheless, there are good prospects of maintaining durable resistance based on major genes. Many known genes for resistance have not yet been exploited in the production of new varieties, and induced mutations can provide new sources of resistance (Gangadharan and Mathur, 1976). Multiline or composite varieties, comprising lines or varieties carrying different resistance genes, have been used experimentally to control rice blast disease in Japan (Toriyama, 1972) and in China (Chiu and Teng, 1975); these may contribute significantly to the control of blast disease in the future.

Maize (Corn)

SOUTHERN CORN LEAF BLIGHT

Maize (*Zea mays*) is attacked by three important leaf blight diseases, caused by different pathogens that have been included in the same genus of the *Fungi Imperfecti*, namely *Helminthosporium*. There has been much discussion about the correct identity of the perfect stages of the three pathogens involved, but *Helminthosporium (Bipolaris) maydis* has now been reclassified as *Cochliobolus heterostrophus*, and *H. turcicum* as *Exserohilum turcicum* or *Setosphaeria turcica*. Northern corn leaf blight, caused by *E. turcicum* (*H. turcicum*), is found in most years in humid areas of the USA Corn Belt. Although an early and heavy attack of *E. turcicum* can reduce the yields of susceptible varieties by more than 60 per cent, the widespread use of resistant varieties has helped greatly to minimize losses caused by the disease (Hughes and Hooker, 1971). This resistance is controlled by major genes and may be race-specific; resistance-breaking races of *E. turcicum* have been reported in India (Payak *et al.*, 1974) but these reports do not seem to have been confirmed. The second leaf blight disease, *Helminthosporium* leaf spot, caused by *H. carbonum*, is only locally important on maize in the USA, and good sources of resistance are available (Hooker, 1975). The third disease, southern corn leaf blight, which is caused by *Cochliobolus heterostrophus* (*H. maydis*), is very widely distributed throughout the world in tropical and subtropical regions. In spite of its widespread distribution, however, *C. heterostrophus* has not usually been a major pathogen of maize. In the USA, for example, southern corn leaf blight was a very minor disease until 1970, because most inbred lines and hybrid varieties possessed good resistance to it (Hooker, 1972). Some susceptible inbred lines and hybrids did, however, occasionally show severe leaf damage caused by *C. heterostrophus* when conditions were very suitable for the development of the disease.

There are at least two races of *C. heterostrophus*, race O and race T. Isolates of race T had been identified in the 1950s and 1960s in many parts of the world including Europe, Africa, South America and the USA (Nelson *et al.*, 1970), but they were not generally common. In 1969 and 1970, however, a very severe epidemic of southern corn leaf blight developed in the USA on varieties derived from lines with a particular type of male-sterile cytoplasm known as Texas cytoplasm. This epidemic was due mainly to the sudden and widespread distribution of race T of *C. heterostrophus* (Ullstrup, 1972).

Two types of genetically controlled resistance to race O had previously been described. Plants exhibiting the first type show relatively fewer lesions than susceptible plants under the same inoculation conditions. Their resistance is expressed quantitatively and is controlled by several genes, which may be additive or complementary in their action (Pate and Harvey, 1954; Hooker, 1972; Lim, 1975a). The second type is qualitative in expression and is inherited as a recessive character (Craig and Fajemisin, 1969), being controlled by a single recessive gene, designated *rh_m*. This type of resistance is manifested as a chlorotic lesion and has given a very effective control of the disease in the field in the USA, although its expression depends on the influence of other genes, the cytoplasm of the host plant and the race of the pathogen (Smith, 1975).

Resistance to race T is controlled both by nuclear and by cytoplasmic genes (Lim, 1975b). Only plants having Texas (T) or related types of cytoplasm for

male sterility are severely attacked by this race, and male-fertile plants and plants with many other male-sterile cytoplasms have shown good resistance to it under field conditions. The Texas cytoplasm was derived from a single male-sterile plant growing in Texas (Rogers and Edwardson, 1952) and this was very widely used in producing hybrid commercial varieties in many countries including the USA, thereby obviating the need to detassel plants manually or by machine in the production of hybrid seed.

The unusual degree of susceptibility to southern corn leaf blight of hybrids with Texas-type cytoplasm was first discovered during the late 1950s in the Philippines (Mercado and Lantican, 1961); this report was confirmed several years later by Villareal and Lantican (1965). Late in the 1969 growing season, abnormally severe symptoms of the disease were reported in maize and sweet corn crops in many northern and central parts of the USA. The causal pathogen was identified as race T of *C. heterostrophus*. The disease reached epidemic proportions in 1970, and losses of grain yield of more than 50 per cent were reported in some southern States (Hooker, 1972). This outbreak apparently originated in Florida in February, spores of the pathogen being carried on southerly winds to start epidemics in Mississippi, Alabama, Georgia and parts of Louisiana by June. Two months later the disease was severe in most of the major maize-growing areas of the USA. This rapid development of southern corn leaf blight in 1970 was attributable, not only to the extreme susceptibility of hybrid varieties carrying Texas cytoplasm, but also to unusually favourable weather conditions for the spread and development of the disease (Moore, 1970). The disease was less severe in 1971 for two main reasons: first, the weather was less conducive to the development of the disease; second, a large proportion of maize crops comprised resistant varieties with 'normal' cytoplasm. Nevertheless, some fields of varieties with Texas cytoplasm were severely damaged by southern corn leaf blight in 1971 in the southern parts of the Corn Belt.

The two races of *C. heterostrophus* differ in several respects. First, race T is highly specific in attacking only plants with Texas-type cytoplasm whereas race O shows no specificity to different cytoplasms. Second, race O is mainly a leaf pathogen whereas race T can attack the leaf, leaf sheath and the ears. Third, race T produces a specific pathotoxin, which inhibits the root growth of plants with Texas cytoplasm but does not greatly affect plants with 'normal' cytoplasm. The pathotoxin, which is apparently a metabolic by-product of the fungus, adversely affects susceptible hosts by increasing the respiration rate, inhibiting normal oxidative phosphorylation reactions and damaging the mitochondria in such a way that essential electron transport systems do not function properly (Miller and Koeppe, 1971; Arntzen *et al.*, 1973; Comstock and Martinson, 1975; Wilson and Apel, 1975; Flavell, 1975; Bednarski, Izawa and Scheffer, 1977). Although race O produces a similar toxin, this is not specific to any particular type of cytoplasm and is produced only in very low concentrations. The pathotoxin produced by race T is, therefore, responsible both for causing disease symptoms on susceptible host plants and for the host-specificity of its effects.

The severity of southern corn leaf blight in different maize lines is partly dependent on maternal effects (Hallauer and Martinson, 1975a, b) and lines with different cytoplasms differ in their reactions to specific isolates of *C. heterostrophus* (Hooker *et al.*, 1970; Julis, Leonard and Moll, 1976). However, the cytoplasmic influence on susceptibility can be modified to some extent by

nuclear genes which have an additive effect (Scott and Futrell, 1975). Thus, plants with Texas cytoplasm carrying chromosomal genes for resistance to race T are not as resistant to this race as plants with normal cytoplasm and the same resistance genes. Factors in Texas cytoplasm also control male sterility, but they are probably not directly associated with susceptibility to *C. heterostrophus* because plants with other male-sterile cytoplasm are not necessarily very susceptible to southern corn leaf blight; in addition, the restoration of pollen fertility to plants with Texas cytoplasm by the gene *Rf*₁ does not affect their disease reaction to race T (Hilty and Josephson, 1971).

MAIZE (CORN) RUST

This disease, which is caused by *Puccinia sorghi*, is common in most countries where maize is extensively cultivated, and can cause severe damage to the leaves and grain yield in susceptible varieties when development of the disease is favoured by periods of high humidity. Yields of fodder maize have been decreased by 50 per cent following artificial inoculation (Mederick and Sackston, 1970). In the USA, maize rust has been kept in check by the use of resistant varieties for more than 70 years and other control measures have usually been unnecessary, although certain fungicides, including zineb, can be very effective. Serious outbreaks of the disease have been reported in other countries, however, including India (Misra, Sinha and Singh, 1969) and parts of the USSR (Shavliashvili and Zedginidze, 1967).

Mains (1931) reported that inbred lines selected from open-pollinated dent varieties of maize differ in resistance to *P. sorghi* and that the resistance appears to be controlled by a single dominant gene. This gene was shown later to be located on chromosome 10 and its locus was designated *Rp*₁. Subsequent work by Hooker and Russell (1962) showed that six resistant inbreds each possess a different dominant gene for resistance to rust, and these seem to be part of either an allelic series at the *Rp*₁ locus or a set of closely linked genes. Several other resistance genes at other loci have been identified (Hooker and Saxena, 1971), including one or more genes on each of chromosomes 4 and 5, and recessive genes at three independent loci (Malm and Hooker, 1962). Saxena and Hooker (1974) showed that resistance gene *Rp*₃ is dominant with one culture of *P. sorghi* but recessive with another, indicating that the resistance which it conditions is race-specific.

The genes described above control resistance that is, apparently, expressed at all stages of the host plant, but these have not been used extensively in plant breeding programmes in the USA (Hooker, 1967). Other types of resistance to maize rust which are not expressed at the seedling stage have been identified, and most maize varieties in the USA show a high degree of adult-plant resistance. In varieties with this resistance, spread of disease in the field is much slower than in susceptible varieties and the former are often described as 'slow rusting'. Although the mechanisms involved are not understood, resistant plants have few pustules and are not severely affected by the disease. This adult-plant resistance is highly heritable, probably being controlled by polygenes (Hooker, 1967), and is apparently non-race-specific (Hooker, 1969), unlike the incompatible reactions that are exhibited at the seedling stage and controlled by major genes.

Seedling resistance seems to be based on hypersensitivity. In susceptible plants *P. sorghi* grows profusely, with little apparent injury to the host cells. In incompatible responses, however, hyphal growth is limited to a necrotic area surrounding the point of penetration, the reaction varying from a sudden lysis of the fungal tissue in contact with host cells, to the death of hyphae which are growing into previously uncolonized areas (Hilu, 1965). The respiration of the host is stimulated by infection, in both compatible and incompatible reactions, by a compound that is either produced directly by the pathogen or induced in the host as a result of infection; the respiration rate may increase so rapidly in incompatible reactions that the host cells are damaged and cannot support growth of the fungus (Van Dyke and Hooker, 1969).

Conclusions – maize diseases

Southern corn leaf blight has already been controlled to a large extent since the epidemics of the early 1970s by avoiding the use of Texas-type cytoplasm in the development of hybrid varieties. Race T is still present in the south-eastern USA, however, and could build up rapidly if there were a return to varieties based on Texas cytoplasm (Leonard, 1977). There is also the danger that new races of *C. heterostrophus* may arise to which other types of cytoplasm might be particularly susceptible. There should, therefore, not be too much reliance in future on any single source of cytoplasm. To avoid this situation, two or more different sources of cytoplasmically inherited male sterility with common restorer genes could be used in developing new hybrid maize varieties, as suggested by Grogan (1971). There are also good prospects for building up additive resistance genes in breeding material; this should help to avoid the production of varieties with extreme susceptibility to southern corn leaf blight, provided that susceptible cytoplasm is not used (Hallauer and Martinson, 1975a, b). Other factors which may help to avoid the possibility of further disease epidemics, such as those experienced in the early 1970s, include the use of chemical gametocides instead of cytoplasmically controlled male sterility to produce hybrid maize varieties; many effective fungicides are now available to augment other methods of controlling southern corn leaf blight.

The epidemics of *C. heterostrophus* in the USA and elsewhere, which followed the widespread use of Texas-type cytoplasm in hybrid varieties, underlines the potential danger in growing large areas of any crop species with varieties with the same or closely related genetic or cytoplasmic derivation. Such cultivars may be universally vulnerable to attack by particular pests and diseases, or to environmental stress. Ullstrup (1972) has justifiably emphasized that diversity must be maintained in both the genetic and cytoplasmic constitution of all important crop species. The considerable genetic variability of *C. heterostrophus* (Yoder and Gracen, 1975) and other pathogens should not be underestimated if we are to avoid future catastrophic epidemics of plant diseases.

Although environmental conditions in the USA Corn Belt are favourable in most years for the development of maize rust, damage from this disease has usually been very slight. Hooker (1967) has attributed this to the prevalence of varieties with broadly based ('general') polygenically controlled, adult-plant resistance to *P. sorghi*. This contrasts with the situation in rust diseases of wheat, where major genes conditioning hypersensitive reactions in seedlings

have been used widely in the breeding of new varieties. Although many new sources of general resistance to maize rust have been found recently (Kim and Brewbaker, 1977), there is an increasing and widespread interest by many plant breeders in the incorporation of major genes for resistance to *P. sorghi* in their breeding material (Payak *et al.*, 1974). Although there are obviously many advantages to the breeder in exploiting these major resistance genes, it is probable that valuable general resistance will be thereby lost unless strict precautions are taken to avoid this. There is no reason to suppose that *P. sorghi* is less variable genetically than other rust fungi and there is, therefore, a serious danger that resistance-breaking races of the pathogen will occur if new varieties do not have an adequate level of general resistance to maize rust. Kim and Brewbaker (1977) suggest that a continued good control of maize rust will be best achieved by combining general and race-specific types of resistance in future varieties.

Potato

LATE BLIGHT

Potato blight is caused by *Phytophthora infestans* and is often described as late blight to distinguish it from early blight, which is caused by the fungus *Alternaria solani*. Potato blight was introduced into Europe from Mexico about 1840, after which it developed rapidly, causing several very serious epidemics including those of 1845 and 1846 in Ireland. The disease is now widespread in most potato-growing countries, but its severity varies from place to place and from season to season according to the prevailing weather conditions. Potato blight spreads particularly quickly in warm, humid conditions. In susceptible varieties the first sign of attack is the appearance of dark-green or brown blotches on the leaves and stems and, in severe infections, the whole of the haulm can become blackened. Spores (conidia) of the causal fungus *Phytophthora infestans* are produced in infected areas, and these are blown or splashed by raindrops to other leaves or plants to cause new infections by means of zoospores which emerge from the conidia. The disease can cause serious losses of yield in susceptible potato varieties in two ways: first by killing the stems and leaves, thereby decreasing the photosynthetic area; second, by direct infection of the tubers by zoospores from conidia that are washed from the haulm into the soil. Most tuber infections occur through the eyes, lenticels or growth cracks (Lapwood, 1977). Discoloured areas develop in infected tubers, which show a greatly increased tendency to rot in storage. Although varieties differ widely in resistance to blight, no variety is completely resistant to all races of the fungus and chemical control methods are very often used to reduce blight damage to an acceptable level.

Salaman (1911) was probably the first to make a detailed study of resistance to blight. He found that certain derivatives of crosses between *Solanum tuberosum* (the cultivated potato) and a wild Mexican hexaploid species, *S. demissum*, were highly resistant to blight. Later work showed that this resistance originated from *S. demissum* and is inherited as a dominant character. Müller (1935) studied blight resistance in the so-called W races of potatoes, which also were derived from *S. demissum*, and suggested that this resistance is controlled by

a series of major (*R*) genes. These *R* genes, which control a hypersensitive reaction of the leaf cells to *P. infestans*, were subsequently shown to be very race-specific, seedlings carrying them being virtually immune to some variants of *P. infestans* but susceptible to others (Black, 1952). An international scheme for the nomenclature of *R* genes controlling hypersensitivity, and of the physiologic races of *P. infestans* that carry corresponding virulence genes, was proposed by Black *et al.* (1953), and this has been generally adopted.

In spite of the fact that races of *P. infestans* that can sporulate on potatoes with the dominant resistance genes R_1 , R_2 , R_3 , R_4 , R_5 and R_6 from *S. demissum* were found many years ago, many plant breeders continued to use these genes extensively in breeding programmes until comparatively recently. It is now generally recognized, however, that breeding for 'field immunity' to blight based on *R* genes is futile (Simmonds, 1970). It is now recommended that Pentland Dell, a variety with the genes R_1 , R_2 and R_3 for blight resistance, should be given the same fungicide treatments to control the disease in the field as a blight-susceptible variety, King Edward (Howard *et al.*, 1970). Several more genes of the *R* type, in addition to genes controlling other kinds of resistance, have been recognized in *S. demissum* and other species of *Solanum* including *S. stoloniferum*. Races of *P. infestans* with complementary virulence genes for these new *R* genes (designated R_5 – R_{11}) are already widespread in the UK although these genes are not present in any common potato variety (Malcolmson, 1969). These races have not, therefore, become widely distributed because of any selective influence of host genotypes with *R* genes. Field populations of *P. infestans* in the UK frequently contain races that are unselected by host resistance, and many of these are virulent towards a wide range of host resistance genes (Shattock *et al.*, 1977). Howard (1970) considers that any genes which control hypersensitivity are unlikely to be useful in controlling blight because of the capability of *P. infestans* to produce new physiologic races quickly.

The resistance of plants with *R* genes is apparently caused by a typical hypersensitivity reaction, in which there is a rapid death of cells that are invaded by the pathogen. The mechanisms that are involved in hypersensitivity to *P. infestans* are complex and are not fully understood. The production of a lignin-like polymer, and associated increases in chlorogenic acid content and phenylalanine ammonia lyase activity, are manifestations of the response of Orion (with resistance gene R_1) to inoculation with race 4 of *P. infestans* (Friend, Reynolds and Aveyard, 1973). The phytoalexins rishitin and lubimin occur in high concentration in incompatible reactions between potato and *P. infestans*, and are probably important in hypersensitive responses (Nakajima, Tomiyama and Kinukawa, 1975). Some workers (e.g. Fehrmann and Dimond, 1967) have reported a positive correlation between peroxidase activity and hypersensitive resistance to blight. The results of Yamamoto and Matsuo (1976) suggest that the DNA of the pathogen may be involved in the recognition of a host plant by *P. infestans* and may induce a hypersensitive response in incompatible combinations. No relationship was found between resistance to blight and the concentrations of the glycoalkaloids solanine and chaconine in the tissues of the host plant (Frank, Wilson and Webb, 1975).

The failure of the hypersensitive type of resistance to give a long-lasting control of late blight in the field has resulted in an increased interest in so-called 'field resistance', which is controlled by polygenes and which may be

non-race-specific. Much of this interest sprang from results obtained by Neiderhauser, Cervantes and Servin (1954) in the Mexican Agricultural Programme of the Rockefeller Foundation. Unfortunately, the presence of *R* genes can seriously interfere with selection for improved field resistance (Black, 1970). Field resistance to blight, which is present in many potato varieties and in certain wild species including *S. demissum* and *S. stoloniferum* (Toxopeus, 1964), is apparently a complex of different types of resistance, the sum of which determines the observed level of resistance to blight in the field (Deshmukh and Howard, 1956; Thurston, 1971). These types include: (1) resistance to infection; (2) inhibition of growth of *P. infestans* in the plant tissues; (3) increased incubation period in the host; and (4) reduced sporulation of the pathogen. Müller and Haigh (1953) sprayed detached leaves with a weak suspension of *P. infestans* zoospores and scored them for extent of disease five days later; the number of infections per unit area on the leaves of different varieties reflected the resistance to blight of intact plants in the field. Umaerus (1963) studied the rate of growth of *P. infestans* in leaves of intact plants of several varieties and found that this was closely correlated with the degree of field resistance shown in natural field infections. The generation time of the pathogen is usually longer and sporulation is less profuse on field-resistant than on susceptible varieties (Lapwood, 1961a), these factors often being related to the rate of mycelial growth of *P. infestans* in the leaf. Potato varieties can apparently express all or none of these four main types of field resistance to late blight, suggesting that these are not under the same genetic control (Hodgson, 1962). Many different methods for detecting varietal differences in field resistance to *P. infestans* in the laboratory or glasshouse have been developed (e.g. Tai and Hodgson, 1975; Piotrowski, 1975; Umaerus and Lihnell, 1976; Malcolmson, 1976). Garcia, Thurston and Tschanz (1977) planted tubers or seedlings in a polyethylene greenhouse about six weeks before they were inoculated with a suspension of 3000 sporangia/ml by means of a hand sprayer. A high humidity was maintained inside the greenhouse by filling it with steam, or by spraying the plants with a fine mist. Although the results of such tests have generally been closely correlated with those of naturally infected field trials, some discrepancies have been reported (Guzman, 1974), and artificial tests are unlikely ever to supplant field evaluation tests completely.

A high level of field resistance is unfortunately often associated with late maturity (Toxopeus, 1964). In a study of the blight resistance of 43 potato varieties, Lapwood (1961b) found that most early varieties are more susceptible than Majestic and that most of the late-maturing varieties are more resistant; hastening the maturity of a late variety by growing it under short-day conditions reduces its field resistance to late blight (Howard, 1970). Nevertheless, Thurston (1971) reports that this association between field resistance to blight and late maturity can be broken, and it should therefore be possible eventually to produce early-maturing resistant varieties.

Although there is no clear-cut association between resistance to late blight in the leaves and the tubers, tuber and leaf resistance occur together in a high proportion of field-resistant varieties and the two characters may be controlled by the same genetic system. There are large varietal differences in tuber resistance: for example, over a period of several years, Majestic had about 1 per cent of infected tubers, King Edward had 2 per cent, Arran Banner had 8 per cent and Gladstone had 10 per cent (Cox and Large, 1960). The popularity of Majestic

as a maincrop variety in the UK has been attributed largely to its high tuber resistance to blight. In early varieties a high level of tuber resistance is less essential because they will normally be harvested before blight is prevalent or damaging.

Satisfactory methods of testing and selecting for resistance of tubers to blight infection have been developed in the field (e.g. Durska, 1975; Howard, Langton and Jellis, 1976). One method uses plots consisting of 16 plants that are surrounded by a single row of blight-inoculated plants of a very susceptible variety; plants are dug in September and the tubers scored for damage caused by blight (Howard *et al.*, 1976). In general, the results of such tests have agreed with those from large-scale field experiments. Attempts have also been made to develop suitable laboratory or glasshouse tests for tuber resistance. For example, Lapwood (1967) grew wound-free tubers of several varieties in peat, sprayed them with a suspension of *P. infestans* spores and recorded the total number of penetrations by the pathogen in each tuber. Low numbers of penetrations, either through the eyes or lenticels, were recorded, and less rotted tissue per infection was observed in tubers of varieties known to have good tuber resistance in the field. The tubers of some resistant varieties developed many primary infections but, as the pathogen did not spread from these, the tubers rotted only slightly. Langton (1972) devised a laboratory test in which resistance of tubers was assessed by comparing the frequency and speed with which *P. infestans* grew through 15 mm diameter cores of tuber tissue below inoculated wounded eyes. This method has the advantage of using only a small number of tubers, and the results obtained generally agree with those from field experiments. Zalewski, Helgeson and Kelman (1974) punctured tubers of different varieties with needles before spraying them with a suspension of *P. infestans* zoospores. After the inoculated tubers had been kept for two weeks at 90 per cent relative humidity, the numbers and extent of infections were recorded for each tuber as a measure of tuber resistance. Walmsley-Woodward and Lewis (1977) have also shown that the field resistance of tubers can be detected in the laboratory by observing the effects of artificially inoculating the eyes and lenticels of tubers. It is unlikely, however, that any single laboratory method will enable the plant breeder to identify and select for all the important components of tuber resistance to blight. Lacey (1966) showed that the distribution of tubers in ridges in the field is mainly an inherited characteristic. Varieties with short stolons have tightly clustered tubers near the soil surface and, as a result, show an unusually high proportion of blighted tubers; conversely, varieties with long stolons have deep and widely spaced tubers with only a small proportion of diseased tubers. Such varietal differences in disease escape are unlikely to be detected in laboratory tests with isolated tubers.

Field resistance seems to be governed by a series of minor genes, with no clear-cut evidence of dominance (Toxopeus, 1959). This contention is supported by Black (1961), and Killick and Malcolmson (1973), who found that progenies of crosses between field-resistant and susceptible plants varied in degree of foliage resistance from fairly resistant to very susceptible. Howard (1970) points out that a polygenic control of field resistance to blight is only to be expected, because the resistance of the foliage is caused by several apparently independent factors. Killick and Malcolmson (1973) found that non-additive effects of specific combining ability were much more important than additive

general combining ability in resistance to blight; epistasis is also a major component of the genetics of field resistance to *P. infestans*.

Although adequate levels of field resistance are present in many potato varieties, even greater levels of such resistance have been reported to occur in some clones of *S. tuberosum* spp. *andigena* in which there are no *R* genes (Simmonds and Malcolmson, 1967). Andigena potatoes have been avoided by most breeders in the past because they have been considered to be too susceptible to blight, but a few of these may prove to be very good sources of non-hypersensitive types of resistance.

WART DISEASE

Wart disease, which is caused by the fungus *Synchytrium endobioticum*, has been considered to be of such potential importance that, for more than 60 years, quarantine regulations have been in force in several parts of the world to prevent its spread within affected countries, and from one country to another (Noble and Glynne, 1970). The disease has been fairly successfully controlled by a combination of legislation and resistant varieties.

The wart fungus attacks leaves, stems, tubers and stolons, but not roots. Infected young developing tubers of susceptible varieties can become very distorted; in older tubers only the buds are infected and these then grow into brown or black wart-like protuberances at, or below, soil level. Sporangia are formed in the warts, and motile zoospores are released from these into the soil to infect other parts of the same or neighbouring tubers.

Synchytrium endobioticum is very common in the Andes region of South America and is now widely distributed in Europe. It has also been found in certain parts of North America and New Zealand. Resistance to wart disease was first recognized in the UK in 1908 by Gough (1920) who confirmed previous reports that some varieties, particularly Snowdrop (also known as Witchhill), remained unaffected by wart even in badly contaminated soil; these were referred to as 'immune' varieties. When it was realized that *S. endobioticum* was very widely distributed in the UK, notification of the disease was made compulsory and the planting of susceptible varieties of potato was prohibited on land where the disease had been observed. Wart disease would probably have remained localized and rare in the UK, even without legislation, as long as the most popular varieties were Victoria, Regent, Champion, Maincrop, Abundance and Bruce, which are resistant to wart. In the early 1900s the only widely grown susceptible variety in the UK was Magnum Bonum, and wart disease became common only when susceptible varieties such as King Edward, President and Arran Chief, became widely grown.

Existing and new varieties were initially tested for reaction to wart disease by planting tubers in contaminated soil and subsequently examining them for warts. Laboratory and glasshouse tests have also been extensively used and these have greatly increased the speed and reliability of testing for resistance. Many of these methods involve planting tubers in soil or potting compost containing sporangia of *S. endobioticum* in rotted or powdered warts (Noble and Glynne, 1970). Glynne (1925) attached young living warts to sprouts of tubers which were then kept damp to encourage liberation of the zoospores and subsequent

infection of the sprouts. Lemmerzahl (1930) modified this method by applying a ring of melted petroleum jelly round the shoot to retain the inoculum; the wart material and ring were removed after an incubation period of several hours, and the tubers were embedded in damp peat for incubation. Warts usually developed on field-susceptible varieties about four weeks after inoculation in these tests. This technique, known as the Glynne-Lemmerzahl method, and other minor modifications of it (e.g. Sharma and Cammack, 1976) are now used in official tests throughout the world for resistance to wart disease. There is usually a good agreement between the results of laboratory and field tests of resistance.

The results of such tests have shown that *S. endobioticum* can infect many varieties which have been regarded previously as immune, and that there is a continuous gradation from the most resistant varieties, in which the fungus soon dies without producing sporangia, to very susceptible varieties, in which numerous warts and sporangia develop (Pratt, 1976). It has been difficult, therefore, to differentiate clearly between susceptible and resistant varieties for statutory purposes. This lack of clear-cut distinction between resistant and susceptible potatoes has also hampered the breeder in deciding which clones should be discarded from breeding material. This is a very important decision, because resistance to wart can be of overriding importance in some countries where wart-susceptible varieties are not accepted for general cultivation.

Lunden (1950) considered that immunity to the most common race of *S. endobioticum* may be controlled either by a single dominant gene, *X*, or by two complementary genes, *Y* and *Z*. There are many different degrees of resistance to wart and the reaction of varieties depends to some extent on the prevailing environmental conditions; the genetics of wart resistance is therefore probably much more complex than this (Howard *et al.*, 1970). As long as one parent of a cross is immune, however, at least 50 per cent of the progeny are usually immune and breeding for resistance is not difficult (Howard, 1970).

In the UK and most countries of western Europe, there is apparently only one race (race 1) of *S. endobioticum*, but several races have been identified in central and Eastern Europe, Russia and Newfoundland (e.g. Olsen and Nelson, 1964). For example, at least eight races have been identified in Germany (Maris, 1961) where most varieties are susceptible to the common races (Langerfeld, 1977); new virulent races have recently been found in the Carpathian zone of the USSR (Yakoleva, 1975) and in Poland (Malec, 1974). However, resistance-breaking races of the wart pathogen have not spread rapidly, presumably because *S. endobioticum* is a soil-borne organism which is not disseminated by airborne spores, and because the movement of seed potatoes that might harbour the disease is controlled by legislation in many countries. In Poland, however, the varieties Sebago and Blunik, which are resistant to race B1 of *S. endobioticum*, became severely attacked by a new race under conditions which favoured infection. Fortunately there are many sources of resistance to *S. endobioticum*, both within potato varieties and in *Andigena* potatoes (i.e. *S. tuberosum* spp. *andigena*) which have not so far been used; these could be introduced into breeding programmes for areas where resistance-breaking races have been a problem. Hybrids between certain *S. tuberosum* varieties and *Andigena* accessions have shown a high level of resistance to a broad spectrum of races of the wart pathogen (Rothacker *et al.*, 1974).

Conclusions and future possibilities

The growing of varieties carrying *R* genes for resistance to blight led quickly to the production of races of *P. infestans* which were capable of attacking them. It has now been realized that further attempts to exploit hypersensitivity controlled by *R* genes would be fruitless, and work on other types of resistance has been intensified. Useful levels of 'field resistance' have been identified in *S. tuberosum* varieties and Andigena potatoes, and field resistance to blight is now an important breeding objective in areas where blight is a potential threat to the potato crop. Although field resistance is a much more difficult character to select for in a breeding programme than hypersensitivity, it should be more durable. Some very old European varieties are still field-resistant to blight after being grown on a large acreage for many years; for example, Champion has shown an acceptable level of field resistance to blight since it was first introduced in Ireland in 1877 (Howard *et al.*, 1970; Thurston, 1971). This suggests that *P. infestans* can only adapt itself to some potato varieties to a limited extent, although Caten (1974) found that variants of a physiologic race of *P. infestans* can become differentially adapted to individual field-resistant varieties on which they are growing in the field. Some erosion of field resistance to blight must therefore be expected, but Caten considered that field resistance is unlikely to break down as rapidly or as completely as resistance controlled by *R* genes. There are therefore good prospects of achieving an effective level of durable field resistance to late blight. This resistance is unlikely to give a complete control of the disease, either on the foliage or in the tubers, and it may sometimes be necessary to apply fungicides even to field-resistant varieties. Fry (1975) has shown that control of late blight by fungicides is much more effective on field-resistant than on susceptible varieties. Integrated control programmes should, therefore, greatly reduce the amount of damage that is caused by this disease.

Wart disease has been successfully controlled in many countries through a combination of legislation and the cultivation of potato varieties with resistance controlled by major genes. Some potato breeders have recently questioned whether too much emphasis is given to wart disease, because susceptible varieties continue to be widely and successfully grown; for example, in 1967 two susceptible varieties, King Edward and Bintje, occupied 16 and 32 per cent of the UK and Netherlands potato acreage respectively, and no extensive outbreaks of wart disease were recorded. Nevertheless, as breeding for resistance to wart disease is not difficult, it may be sensible to maintain the objective of avoiding the introduction of wart-susceptible potato varieties. It is also important to retain, and to extend if necessary, the legislative measures for containing the disease which have played such an important part in controlling wart disease so effectively for many years in the UK and certain other countries.

Coffee**LEAF (ORANGE) RUST**

Coffee is one of the most important products in international trade, and the economies of more than 50 countries depend largely on coffee production (Rodrigues, Bettencourt and Rijo, 1975).

Crops of the tetraploid species, *Coffea arabica*, provide about two-thirds of the world's coffee, mainly in South America. Most of the remaining one-third is produced from Robusta, Uganda and Quillou coffees, which are types of a diploid species, *Coffea canephora*, grown mainly in Africa. None of the other species of *Coffea*, all of which are diploid, are of significant economic importance.

Coffee rust, caused by the fungus *Hemileia vastatrix*, is the most serious disease of coffee, and epidemics of rust in several parts of the world have been responsible for acute shortages of coffee on several occasions during the past century. This disease was first identified in Ceylon (now called Sri Lanka) in 1869, where it became so widespread and damaging during the next 20 years that it was no longer economic to grow coffee on the island. In the first two or three decades of this century, coffee rust spread to most of the main African and Indian coffee-growing areas, but the disease did not become established in Angola until 1966. Coffee rust spread to Brazil, perhaps by means of airborne spores carried across the Atlantic Ocean from Angola to South America, where it was first identified in 1970. The disease has since spread to many other parts of South America including Paraguay and Argentina (Schieber, 1975).

In spite of attempts to control coffee rust with fungicides, particularly those which contain copper, the disease has continued to spread and damage the crop, with severe infections resulting in defoliation. The timing of fungicide applications is particularly crucial and, in years with unusual weather patterns, significant defoliation can occur even in fungicide-treated crops (Waller, 1971). Although improved methods of disease control, including the use of new fungicides and new growing techniques, are being widely used (Schieber, 1975), resistant varieties are playing an increasingly important part in controlling coffee rust throughout the world.

The search for sources of resistance to *H. vastatrix* has been carried out in several countries, but particularly in Portugal at the Coffee Rust Research Center (D'Oliveira, 1965), in Brazil (Rodrigues *et al.*, 1975) and in East Africa (Rayner, 1960). Experiments carried out in Portugal during the 1950s showed that all the *C. arabica* types that were grown extensively in South America at that time were very susceptible to many common isolates of *H. vastatrix* (D'Oliveira, 1965). Many *C. arabica* types from other parts of the world have since been tested for resistance; each type has been resistant to some isolates of the pathogen and susceptible to others (Rodrigues *et al.*, 1975a). The resistance of *C. arabica* is therefore highly race-specific and the sudden 'loss' of rust resistance shown by many varieties in Asia and Africa in the past century can be attributed to the spread of resistance-breaking variants of *H. vastatrix*. More than 30 physiologic races of the pathogen have been distinguished using a series of 17 differential host genotypes. One of these races, designated Race II, is particularly widespread, having been found in most of the important coffee-growing areas of America, Asia and Africa. Five dominant genes for resistance to *H. vastatrix* have been identified in *C. arabica*, and these are designated *SH*₁, *SH*₂, *SH*₃, *SH*₄ and *SH*₅. Each of these genes conditions resistance to specific races of the pathogen.

Some of the diploid species of *Coffea*, particularly *C. iberica* and *C. canephora*, are much less damaged by rust than *C. arabica*, although populations of each species apparently contain both fully resistant and fully susceptible plants. There should, therefore, be considerable scope for improving the resistance of these

species by discarding the susceptible plants from the populations. Unfortunately, these diploid species give coffee beans of lower quality than the best types of *C. arabica* and are unlikely, for this reason, to become widely grown in South America. Although resistant hybrids between *C. arabica* and resistant diploid species have been produced, the triploid hybrids have usually been unproductive because of sterility resulting in a large proportion of unfilled beans. However, Hybrido de Timor, which is a natural hybrid between *Coffea arabica* and *C. canephora*, is grown commercially and has shown good resistance to a wide range of *H. vastatrix* races. In Brazil, progenies of crosses between *C. arabica* and *C. canephora* have been shown to contain plants that are resistant to all known races (Monaco, 1972).

Work on the production of resistant varieties has been carried out in many countries including India (Vishveshwara and Govindarajan, 1970), Tanzania (Fernie, 1964), Kenya (Firman and Hanger, 1963), Brazil (Monaco and Carvalho, 1975) and Colombia (Castillo, Lopez and Torres, 1972). Many new hybrids between varieties of *C. arabica* from many parts of the world have been produced at the Coffee Rust Research Center in Portugal, and the most promising material has been released to plant breeders throughout the world. Much of this resistant material, particularly that from Ethiopia, India and Kenya, is too unproductive in South America for it to be of direct use there (Monaco, 1972). Nevertheless, this material is potentially useful as a source of resistance in developing locally adapted, high-yielding, high-quality varieties.

Most breeding programmes have involved the use of the five major resistance genes in the series SH_1 – SH_5 which condition hypersensitivity to the pathogen in *C. arabica*. This hypersensitivity is apparently associated with an accumulation of antifungal compounds in diffusates from leaves that have been inoculated with incompatible races of *H. vastatrix*. These compounds inhibit both the germination of spores of the pathogen and the growth of the germ tubes. In compatible reactions, however, diffusates from inoculated leaves do not contain such antifungal substances (Rodrigues, Medeiros and Lewis, 1975). This type of resistance reaction is similar to those which occur in major-gene-controlled hypersensitivity in many other race-specific host–parasite relationships. There is very little published information about the nature of resistance to coffee rust in other species of *Coffea*, but mechanisms that are different from those controlled by *SH* genes seem to be implicated. Interspecific differences in concentrations of chlorogenic acid in the plant tissues have been found, but no causal relationship has been established between chlorogenic acid and resistance.

Experience with coffee rust epidemics in three continents suggests that the use of the *SH* major resistance genes, whether they are used singly or in combination, is unlikely to provide a permanent solution to the problem of controlling *H. vastatrix*. New races of the pathogen in the major coffee-growing regions of Brazil are already a threat to several varieties that were resistant to coffee rust when they were first introduced a few years ago (Schieber, 1972; 1975). It is important, therefore, for plant breeders to look for and to use types of resistance that are more durable than those controlled by the *SH* genes. Durable resistance has apparently been found in *C. arabica* varieties in Angola and Brazil (Schieber, 1975) and a search for other resistant coffees in Ethiopia and the Sudan may yield other sources of durable resistance. Differences in horizontal resistance to *H. vastatrix*, involving reduction of infection rate, lesion growth and sporulation and also of tolerance, have been detected in *C. arabica* and

C. canephora (Monaco, 1977). The resistance to coffee rust in many varieties of *C. canephora* seems to be sufficiently effective and durable to avoid catastrophic epidemics. For this reason, further attempts should be made to transfer this resistance from *C. canephora* to high-yielding and good quality *C. arabica* types.

Sugar beet

CERCOSPORA LEAF SPOT

Cercospora leaf spot caused by *Cercospora beticola* is a widespread and damaging disease of sugar beet which occurs in parts of Europe, Africa, Asia and North and South America. It is particularly important in areas with warm and humid summers, such as southern Europe, eastern parts of North America, and Japan (Bleiholder and Weltzien, 1972). The symptoms are typical of leaf spot diseases, consisting of circular brown lesions about 5 mm in diameter, divisible into two distinct zones – a central grey zone in which the host tissues are killed, and a surrounding brownish zone where the host cells are damaged but not killed. Severe infections, in which the spots coalesce and leaves are killed prematurely, can cause the loss of more than 50 per cent of the potential sugar yield in susceptible varieties. Fungicide applications can greatly increase the sugar yield of susceptible varieties in severe epidemics, but complete control of leaf spot cannot be achieved under such conditions even with repeated applications of suitable chemicals (Coe, 1968). Systemic fungicides, particularly benzimidazole derivatives, have been widely used to control *Cercospora* leaf spot, but many of these are no longer effective because fungicide-tolerant variants of the pathogen have become widespread (Ruppel and Scott, 1974; Dovas, Sylakakis and Georgopoulos, 1976). Resistant varieties have been developed in several countries and these have given a fairly good control of leaf spot in the field. Losses of sugar yield of about 20 per cent in resistant varieties, compared with more than 50 per cent in susceptible varieties, have been reported in severe field infections; leaf spot can therefore cause considerable yield losses even in resistant varieties, and further improvements in the level of resistance are desirable.

Resistance to leaf spot was first found in a small number of sugar beet lines, derived from European varieties, out of many tested by the United States Department of Agriculture during the 1930s and 1940s. From these and similar lines, a number of resistant varieties were developed by a complex system of mass selection, inbreeding and hybridization; plants which showed smaller and fewer *Cercospora* spots per unit area of leaf than others, were selected and resistant family lines were developed from them (Stewart, 1948). Considerable improvements in resistance to *Cercospora* were achieved by these and similar methods in the USA (e.g. Oldemeyer and Zielke, 1967), Europe (Barocka, 1969) and Japan (Hosokawa and Saito, 1965); several resistant monogerm varieties are now being grown in these areas. Some breeders (e.g. Koch, 1970) have reported that resistant varieties are often low-yielding types, but some high-yielding resistant varieties have been developed and there is apparently no direct correlation between high resistance and low yield.

Attempts have been made to use *Beta vulgaris* s.sp. *maritima* and other species of *Beta* as sources of higher levels of resistance than are available in sugar beet varieties. Some progenies of crosses between sugar beet and leaf spot-resistant accessions of *B. maritima* have expressed a moderate to high level of resistance to leaf spot, combined with good agronomic characteristics (Bilgen *et al.*, 1969). However, these plants are not immune to *C. beticola* and can still be damaged by leaf spot; sources of immunity have therefore been sought in other species of *Beta*. Coons (1954) reported that the *Patellares* section of *Beta* contains several species which are almost immune to both *Cercospora* and curly top virus; these include *B. patellaris*, *B. webbiana* and *B. procumbens*. Later work in Germany (Bornscheuer and Schlösser, 1961) and Poland (Osińska, 1968) has confirmed the resistance of several species in the *Patellares* section, and has shown that *B. atriplicifolia*, *B. trigyna* and *B. lomatogona* are also resistant. Although it is difficult to hybridize these species with *B. vulgaris*, it might be possible eventually to introduce valuable resistance genes from species in the *Patellares* group into sugar beet. Significant progress has already been made in producing intraspecific hybrids with acceptable agronomic characteristics (Savitsky and Price, 1965; Savitsky, 1975).

Hosokawa and Saito (1965) recognized three components of resistance to *C. beticola*, namely genetic, environmental and genetic-environmental interaction, and three corresponding components of virulence in the pathogen; in both host and pathogen the genetic component is the most important. Saito (1966) found that there is a continuous variation in the expression of resistance in progenies of crosses between resistant and susceptible plants, and concluded that resistance is governed by polygenes. Kikindonov (1967) also found that resistance to *C. beticola* is not simply inherited, because resistance is dominant in some crosses between resistant and susceptible varieties, and recessive in others; he could not detect any maternal influence on the inheritance of resistance, nor is resistance apparently associated with any particular ploidy level. Smith and Gaskill (1970) reported that resistance to *Cercospora* leaf spot in sugar beet lines developed in Colorado is inherited quantitatively, being controlled by at least four or five pairs of genes. Later experiments with breeding material in artificially induced field epidemics showed that additive gene effects are important in the inheritance of resistance (Smith and Ruppel, 1974). Lewellen and Whitney (1976) have confirmed the existence of quantitatively inherited resistance to *C. beticola* in California and reported the presence of a quite different type of resistance in GW359 and US201. A single dominant gene, *Cb*, conditions a hypersensitive 'fleck' reaction to race C2 of *C. beticola* but not to race C1. Isolates of *C. beticola* collected in California, that are avirulent on sugar breeding lines from Colorado, were designated race C2 by Whitney and Lewellen (1974); the term 'race C1' was applied to Californian isolates of the pathogen that could attack all the plants against which they were tested.

Many races of *C. beticola* have been differentiated either by their pathogenicity, by their characteristics in culture or by their ability to tolerate fungicides. The pathogen shows considerable genetic variability arising mainly from somatic mutations; such mutations can readily be induced by exposing *C. beticola* conidia to ultra-violet light for a few minutes (Brillová, 1976). Schlösser and Koch (1957) compared the response of susceptible and resistant sugar beet varieties in the field to a range of naturally occurring *C. beticola* isolates collected from several parts of Europe. At the start of the growing

season, Spanish and Italian isolates produced the most severe symptoms, and those from Germany, France and Canada the mildest symptoms. One isolate from Germany, however, caused more damage on susceptible varieties than any of the other *C. beticola* isolates tested, but less damage than other isolates on resistant varieties; this suggested that the resistance in these varieties is partly race-specific. In Hungary, several physiologic races have been differentiated by tests involving a number of beet varieties (Hetzer and Kiss, 1964), and three distinct races have been distinguished in Israel according to the number of lesions produced by them on leaves of a set of sugar beet varieties (Solel and Wahl, 1971). The race specificity of some kinds of resistance to *Cercospora* is emphasized by the fact that an American sugar beet breeding line, which is resistant in the USA, has been found to be highly susceptible in Hungary (Calvert *et al.*, 1970).

The nature of resistance to *Cercospora* has been studied extensively in several countries, but no completely satisfactory explanation of resistance has yet been put forward, perhaps because there seem to be several different types. Stewart (1948) reported that fewer and smaller spots develop on resistant plants, and that resistance is associated with a delay in tissue breakdown in and around the spots. Osińska (1970) found that *C. beticola* grows faster and produces more conidia in leaves of susceptible varieties than in those of resistant varieties. The size and density of stomata on the leaf surface are probably not important factors in resistance, because resistant and susceptible varieties usually have similar numbers of stomata of the same size (Schlösser, 1969). There is probably no inhibition of spore germination on the leaf surface, although fewer germ tubes of *C. beticola* successfully complete the infection process in resistant plants (Solel and Mintz, 1971). Schlösser (1969) has suggested that part of the resistance to *C. beticola* may be caused by the presence of fungitoxic saponins in the leaves. Several workers have reported that leaves of resistant plants contain higher concentrations of certain polyphenols and show greater polyphenol oxidase activity than susceptible plants (e.g. Trzebiński, 1961). A high concentration of a polyphenol compound, 3-hydroxytyramine, is apparently correlated with resistance to leaf spot (Maag *et al.*, 1967); this compound, which is present in high concentrations in both healthy and diseased leaves of resistant plants, is toxic to *C. beticola* in pure culture, but its importance in conferring resistance in the intact plant has not been conclusively demonstrated. The concentrations and activity of peroxidase and orthodiphenol oxidase increase to a greater extent in resistant plants in the first six days after infection than in susceptible plants (Rautela and Payne, 1970); this supports the contention that phenolic compounds are implicated in resistance.

Highly resistant sugar beet varieties, when inoculated with *C. beticola*, produce greater quantities of an isoflavone, betavulgaria, which has many of the properties of phytoalexins and is fungitoxic, than do susceptible varieties (Johnson *et al.*, 1976). Hecker *et al.* (1975) have reported that the concentration of the amino acid dihydroxyphenylalanine is higher in resistant than in susceptible plants at all stages of growth; conversely, there is less L-glutamic acid in resistant plants. These differences in amino acid concentrations have been used to predict the level of resistance to *C. beticola* in different breeding lines.

A non-specific toxin, which is phytotoxic and bacteriostatic (Balis and Payne, 1971), is produced *in vitro* by *C. beticola*; if this toxin is infiltrated at low concentrations into sugar beet leaves, it causes necrotic spots similar to

those seen on infected host plants (Schlösser, 1971). The amount of toxin produced by different isolates of the fungus seems to be correlated with their pathogenicity on sugar beet. Resistant sugar beet may be able to tolerate higher concentrations of this toxin than susceptible plants and may thereby suffer less damage from leaf spot infection.

DOWNY MILDEW

Downy mildew (*Peronospora farinosa* f.sp. *betae*) affects sugar beet, red beet, fodder beet and mangolds in many parts of the world, particularly those with cool, humid climates. Young leaves of infected plants become chlorotic, thickened, brittle and curled, the margins being rolled under and inwards. The under surface of such leaves usually bears a purple-grey covering of mycelium and conidiophores. The conidia are carried in the wind to the leaves of other plants and, if conditions are sufficiently cool and humid, the spores germinate and enter the leaf through the stomata. Susceptible plants that are infected when young can lose more than 40 per cent of their potential yield of sugar (Leach, 1945). Infection decreases the weight, sugar content and juice purity of the roots. The disease is difficult to control effectively with fungicides, and the avoidance of susceptible varieties has played a major part in several countries in reducing economic damage by the disease to an acceptable level (Dixon, 1976). For example, the replacement of susceptible varieties in Yugoslavia with more resistant varieties has led to a significant reduction in the importance of downy mildew (Kovačević *et al.*, 1970).

Matić (1970), in field experiments, compared the resistance to downy mildew of several varieties from many parts of the world, and found that varieties from Yugoslavia, Bohemia and North America are generally more susceptible to *P. farinosa* than are those from humid areas, including the UK and Ireland. It appears, therefore, that varieties that have been developed in areas where downy mildew is common are, to some degree, resistant, presumably as a result of intentional or fortuitous selection for resistance; badly diseased plants would be discarded by the breeder and, in any case, would probably not contribute significant amounts of either pollen or seed in seed multiplication plots. Most sugar beet varieties have had sufficient 'field' resistance to avoid catastrophic outbreaks of downy mildew under most conditions. This partial resistance to *P. farinosa*, which is usually expressed more strongly in polyploid than diploid varieties (Kuzicheva, 1969; Willey, 1969), has been durable and no problems with resistance-breaking races of the pathogen have been reported.

The first deliberate attempt to select for resistance to downy mildew appears to have been made in the early 1940s by Leach (1945) in California. He raised seedlings of a range of varieties in the glasshouse, inoculated them by spraying with a suspension of *P. farinosa* conidia in water, and discarded plants which showed severe downy mildew symptoms. By breeding from the remaining plants, Leach developed an improved downy mildew-resistant strain of US33. McFarlane (1952) used a similar technique to screen large numbers of plants from different lines and varieties, and confirmed that progenies of plants selected for resistance in the glasshouse showed improved resistance when tested in the field. In the UK, however, Russell (1968a) showed that the resistance of sugar beet varieties determined by glasshouse tests of seedlings was not always a

reliable guide to their resistance under field conditions; in addition, the results of seedling tests in the field often differed from those of mature plants, suggesting that the resistance of certain stocks, relative to each other, can vary at different growth stages. For example, young plants of Maris Vanguard were more susceptible to downy mildew than those of Hilleshog N at the beginning of the season, but this position was reversed two months later.

Although glasshouse tests can be useful in selecting and testing seedlings for resistance to downy mildew (Brown, 1977), it is more satisfactory to expose plants to an artificially induced epidemic of the disease throughout the growing season. This method has been used with success in the UK; plants which did not show severe downy mildew symptoms during a complete growing season were selected for seed production (Byford, 1969; Russell, 1969; Willey, 1969). Progenies of plants that had been so selected were more resistant to downy mildew than were the parent stocks in field tests; fewer plants became infected, and those that did, showed milder symptoms than did plants of the unselected parent lines. Only one generation of such selection often resulted in a significant improvement in 'field' resistance. Several components of resistance are probably involved in this field resistance, including resistance to germination of conidia on the leaf surface, a tendency to escape infection, resistance to growth of *P. farinosa* in the leaf tissues, a prolonged generation time, decreased sporulation and tolerance (Russell, 1972). The concentration of sugars in beet leaves seems to be an important factor in some of these components (Russell, 1968b; Russell and Barford, 1971). Individual sugar beet genotypes may exhibit none or several of these resistance components, each of which seems to be inherited independently of the others and appears to be controlled by several genes.

Resistance to downy mildew based on a hypersensitive response of epidermal leaf cells to *P. farinosa* was discovered by Russell (1969) in an American inbred



Figure 4.7 Major-gene resistance to downy mildew in sugar beet. Rows from left to right:— Line G (susceptible) not inoculated; Line G inoculated with *Peronospora farinosa* f.sp. *betae* race O; Line F (resistant) not inoculated; Line F inoculated with *P. farinosa* race O. (By courtesy of Plant Breeding Institute, Cambridge)

line, FC63-9058 (Figure 4.7). This form of resistance, which is controlled by a single dominant gene, gave an excellent control of the disease in field and glasshouse experiments on plants of all ages, for three years after its discovery in 1967. Deliberate attempts were made to encourage the development and selection of new races of *P. farinosa* which could attack this line, by subjecting seedlings to natural and artificial inoculation with the pathogen, over a period of three years. Conidia of *P. farinosa* were observed on this line for the first time in 1970 in a glasshouse inoculation test. This new race (race 1) was able to infect other plants of line FC63-9058, whereas other isolates (race 0) of the pathogen

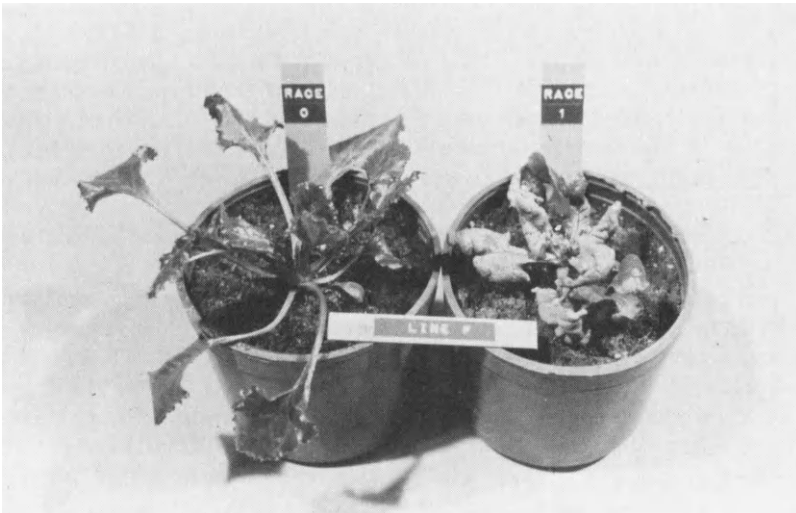


Figure 4.8 The resistance of sugar beet Line F (FC63-9058) is race-specific because this line is resistant to most isolates of *Peronospora farinosa* f.sp. *betae* (race 0) but is very susceptible to race 1. (By courtesy of Plant Breeding Institute, Cambridge)

were not (Figure 4.8). This showed that this major gene, which conditions a typical hypersensitivity 'fleck' reaction, is race-specific, and emphasized the potential danger of using this type of resistance in breeding material unless it is accompanied by other non-race-specific kinds of resistance.

Tomato

FUSARIUM WILT

Fusarium wilt (*Fusarium oxysporum* f.sp. *lycopersici*) is a serious disease of tomato (*Lycopersicon esculentum* L.) in most areas of the world where there are high soil temperatures during the growing season. The fungus enters the roots from the soil and infects the vascular system, in which it causes a brown discoloration. In susceptible plants the external symptoms are yellowing and wilting of the leaves, starting at the base of the plant and progressing upwards; this is apparently caused by a specific polypeptide toxin, produced by the

pathogen. The disease causes a large increase in resistance to water flow in the xylem and this induces wilting, particularly when it occurs in the petioles (Duniway, 1971). Although excellent control of *Fusarium* wilt can be achieved by soil drenches of some systemic fungicides, such as benomyl, wilt-resistant varieties have, until recently, been almost the only method of controlling the disease (Walter, 1967; Walker, 1971; Crill *et al.*, 1972).

Several varieties, including Rutgers and Marglobe, with moderate levels of wilt resistance, were developed in the USA before 1939, but these often succumbed to the disease when conditions were particularly favourable for its development (Caldwell, 1939). Sources of greater resistance were therefore sought by plant breeders. Bohn and Tucker (1939) identified a single dominant gene in *Lycopersicon pimpinellifolium* (Accession 160) which conferred immunity to *Fusarium*. This gene, designated *I*, was found to be linked with a gametophyte factor, *X*. This factor and the presence of modifier genes are responsible for significant deviations from the ratios of resistant and susceptible plants in segregating progenies which would have been expected from a monogenic control of resistance (Kedar, Retig and Katan, 1967; Honma and Vriesenga, 1972).

Hybridization between tomato lines and *L. pimpinellifolium* formed the basis of a wilt-resistant variety, Pan America, which was released in the USA in 1941 (Porte and Walker, 1941). Many other varieties carrying this *I* gene for resistance were subsequently produced. Although the early varieties were unsatisfactory in many respects, varieties with this resistance gene enabled ten successive crops of tomato to be produced on sand land in Florida where two crops would have been the limit with other more susceptible varieties because of a build-up of *F. oxysporum* in the soil (Walter, 1967). However, within four years of the introduction of Pan America, a variant of *F. oxysporum* f.sp. *lycopersici* was found which could attack tomato varieties carrying the *I* resistance gene (Alexander and Tucker, 1945). This variant (race 2) did not cause concern, however, until the early 1960s when it damaged many previously resistant varieties in Florida; race 2 is also common elsewhere, including Israel (Katan and Ausher, 1974). Alexander and Hoover (1955) had already shown that genes conditioning resistance to race 2 are present in wild species of *Lycopersicon*. Stall and Walter (1965) found a single dominant gene which controlled resistance to race 2 in a derivative of a cross between a tomato variety and a *L. pimpinellifolium* line (PI 126915); this source of resistance has since been exploited in several varieties. For example, Walter, with resistance to race 2, was grown on more than 75 per cent of the tomato acreage in Florida during the early 1970s following severe attacks by race 2 on some previously resistant varieties.

The gene which conditions resistance to race 2 is apparently independent of the *I* gene which controls resistance to race 1 (Cirulli and Alexander, 1966). Another race of the pathogen (race 3), which can attack varieties that carry these two resistance genes, has also been found (Alexander and Hoover, 1955; Tokeshi, Galli and Kurozawa, 1966) but race 1 is still the most widely distributed and economically important race of *F. oxysporum* f.sp. *lycopersici*. Varieties which carry the *I* resistance gene are therefore still making an important contribution to the control of *Fusarium* wilt.

The nomenclature of pathogenic races of *F. oxysporum* f.sp. *lycopersici* has been a subject of much recent discussion. Gabe (1975) suggested that a

system of nomenclature, similar to that of Black *et al.* (1953) to classify races of *Phytophthora infestans*, should be adopted for the tomato wilt pathogen. Under this system, race 0 would be virulent only on cultivars possessing no *I* resistance genes; the gene *I-1* would protect against race 0 but not against race 1; gene *I-2* would protect against both races 0 and 1. This system would accord with internationally accepted systems for variants of many pathogens, including races of *Fulvia (Cladosporium) fulva* (tomato leaf mould) and strains of tobacco mosaic virus (tomato mosaic). This logical suggestion is unlikely to be widely accepted, however, because the original system is so well entrenched.

Although the mechanisms of resistance to wilt have been intensively studied for more than 50 years, our understanding of resistance is still very incomplete. Resistance does not appear to be caused by the physical restriction of the fungus through the vascular system of the host (Mace, Veech and Hammerschlag, 1971) and specific biochemical reactions between host and parasite are probably involved (Beckman, 1966; Walter, 1967). An inhibitor of *F. oxysporum*, probably α -tomatine, occurs in roots and stems of infected tomato plants (Langcake, Drysdale and Smith, 1972; Drysdale and Langcake, 1973), but its importance in resistance is unknown. Tissues of susceptible plants show a greater increase in oxidase activity and have a higher protein content after infection than do resistant plants. Resistant plants show an increase in ribonuclease activity after infection, whereas in susceptible plants there is a decrease in activity: this increased activity may help to protect the host against the nucleic acid of the pathogen (Grzelińska, 1969). Toxins present in the xylem vessels of infected plants suppress the growth of *Fusarium oxysporum* and may contribute to resistance (Stromber and Corden, 1977). The importance of the various resistance mechanisms is not known and more information about the nature of different types of resistance to *Fusarium* wilt is urgently needed. Such information will be difficult to obtain, however, because the expression of even apparently simple types of resistance can be affected by many factors, including the concentration of inoculum used, the age of the host plant and environmental conditions (Alon, Katan and Kedar, 1974).

LEAF MOULD

Leaf mould is one of the most widespread and damaging diseases of tomatoes grown under glass, but it can also attack outdoor crops in cool, wet seasons (Manning and Cox, 1973). The causal fungus, *Fulvia fulva* (also known as *Cladosporium fulvum*) produces a mat of mycelium and conidiophores on the undersurface of leaves, and much of the foliage can be killed in severe attacks, with a consequent loss of fruit yield. Although the disease can be controlled by appropriate fungicides, considerable effort has been devoted to breeding resistant varieties in many countries (e.g. Meszoly, Hodcsy and Szurke, 1965; Walter, 1967; Lemeni, 1969).

During the early 1930s all available tomato varieties were screened for resistance to leaf mould (Langford, 1937); one of these, Stirling Castle, was found to be resistant to some isolates of *F. fulva* but susceptible to others. This resistance was later shown to be controlled by a single dominant gene, designated *cf-1*; this gene has since been used in many breeding programmes and is present in

several varieties including Leaf Mould Resister and V473 (Kooistra, 1964). Vetomold, with resistance conditioned by a different gene (*cf-2*), derived from *Lycopersicon pimpinellifolium*, was introduced in Canada and parts of western Europe in 1939; soon after its introduction, however, it was attacked by a new race of *F. fulva*. Another variety, V121, which carries a third dominant resistance gene (*cf-3*), also from *L. pimpinellifolium*, was introduced in 1941 and was soon attacked by a race of the pathogen with matching virulence. Many other varieties with these resistance genes were also badly damaged by leaf mould (Kooistra, 1964).

Several wild species of *Lycopersicon*, including *L. minutum* and *L. esculentum* var. *minor*, are immune to *F. fulva* (Leski, 1970). Single dominant genes that condition apparent immunity to leaf mould are also present in *L. hirsutum* var. *glabratum* and *L. peruvianum*; these species carry the gene *cf-4*, which is located on chromosome 1, probably near the *cf-1* locus, and other genes that confer a lower level of expression of resistance (Kerr and Bailey, 1964). The variety Vagabond, with resistance derived from *L. hirsutum*, was first marketed in Canada in 1954 and it has been extensively used as a source of resistance to leaf mould in breeding other varieties. In 1963 many hybrids derived from Vagabond were attacked by new variants of *F. fulva*, but Vagabond did not itself become infected at that time, presumably because this variety carried other resistance genes in addition to *cf-4*. However, Vagabond was attacked by a new race of the pathogen in 1968.

The value of resistant tomato varieties in controlling leaf mould has, therefore, been much reduced by the development of resistance-breaking races of *F. fulva* (Day, 1954). Seven races had been isolated in Canada by 1950, Vetomold being susceptible to races 5, 6, 7 and 8. Stirling Castle was susceptible to races 2, 6 and 8, and the Vineland strain of *L. pimpinellifolium* to races 6 and 7; however some accessions of *L. hirsutum* were resistant to all known races. Hubbeling (1968) reported that some variants of *F. fulva* could overcome the resistance of Vagabond (genes *cf-2* and *cf-4*) and of Vantage and Vinequeen (genes *cf-2*, *cf-3* and *cf-4*).

At least 12 races of *F. fulva* have now been identified by their reaction on a set of differential tomato varieties (Kerr, Patrick and Bailey, 1971). For example, Purdue 135 is resistant to races 1–9 and 11 but not to races 10 or 12 (Figure 4.9). Several tomato lines, including PI 124161 and PI 126915, carry genes which condition a high level of resistance to races 10 and 11 and a *L. hirsutum* line (PI 199381) is immune to race 11 but not to race 10. A gene (*cf-5*) which gives immunity to races 6, 10, 11 and 12 has been found in another accession, PI 187002 (Kerr *et al.*, 1971) but a new race from Belgium has overcome this resistance (Boukema, 1977). Many of these races have combined virulence to more than one resistance gene and are very widely distributed (e.g. Vlasova and Garan'ko, 1975; Boukema and Garretsen, 1975).

Although the nature of resistance to leaf mould is not fully understood, resistance conditioned by major *cf* genes seems to involve a hypersensitive reaction between specific host genotypes and incompatible variants of *F. fulva*. Susceptible varieties that carry no major resistance genes are extensively colonized by the pathogen, but there is little visible damage until the onset of sporulation. In Vinequeen, which carries at least two major resistance genes, development of *F. fulva* is restricted to a few mesophyll cells and there is a massive deposition of callose in affected cells (Lazarovits and Higgins, 1976 a, b).



Figure 4.9 Race-specific resistance of tomato variety Purdue 135 to leaf mould, caused by *Fulvia fulva* (*Cladosporium fulvum*). (a) Incompatible reaction with race 11 of the pathogen. (b) Compatible reaction with race 12. (By courtesy of Dr Lazarovits, Agriculture Canada, London, Ontario, Canada)

In incompatible reactions, toxins from the pathogen apparently damage cell membranes so that components can leak out of the host cells (Van Dijkman and Kaars Sijpesteijn, 1973). This interaction between specific fungal products and specific receptors in the host plasma membrane may lead to a hypersensitive reaction; if this is so, the receptors in the host cells are controlled by the resistance genes *cf-1* to *cf-5* and the production of specific toxins by the fungus is controlled by corresponding virulence genes.

Factors other than hypersensitivity seem to be involved in some types of resistance to leaf mould. For example, certain resistant tomato varieties and *Lycopersicon* species are reported to contain unusually low concentrations of sugars and the carbon:nitrogen ratio may be a useful guide to the degree of

leaf mould resistance in different tomato lines (Bailey and Lowther, 1962). Low concentrations of certain free amino acids are also correlated with resistance to leaf mould in some tomatoes (Lowther, 1964).

Future possibilities

Although major-gene resistance to *Fusarium* wilt derived from *L. pimpinellifolium* is race-specific, its use in tomato varieties has given quite a good control of the disease in many parts of the world. New races of the pathogen have been identified, but these have not apparently caused great or widespread damage in resistant varieties. This may be partly because *Fusarium* wilt is a soil-borne disease and new races of such pathogens usually spread only slowly. It is possible also that many of the varieties which carry the *I* resistance gene have additional resistance of the type that has been found in certain tomato varieties produced before 1939; Very little is known about the race specificity or genetics of the partial resistance of such varieties, which include Rutgers and Marglobe; however, its presence in some varieties which carry the *I* gene may have contributed to the durability of major-gene resistance. Many current varieties are not as resistant to *Fusarium* wilt as earlier varieties which carry the same *I* genes for resistance, suggesting that some genes conferring partial resistance or 'tolerance' may have been lost (Jones and Crill, 1974).

Several tomato varieties recently produced in the USA have resistance to *Fusarium* wilt combined with high yield, good quality and resistance to other diseases. For example, Henderson and Jenkins (1971) reported that varieties have been developed in North Carolina with resistance to *Fusarium* wilt and bacterial wilt (see page 190). In Florida, genes for resistance to *Fusarium* wilt and at least six other fungus and virus diseases have been incorporated into tomato lines of acceptable yield and quality (Crill, Burgis and Strobel, 1971). Varieties with resistance to race 1 of *F. oxysporum* f.sp. *lycopersici* and *Septoria lycopersici* have been marketed in Australia (Sumeghy, 1975), and varieties with resistance to races 1 and 2 of *F. oxysporum* f.sp. *lycopersici*, to *Verticillium dahliae* and to nematode pests have been developed in France (Laterrot, 1972).

The resistance to *Fusarium* wilt in these varieties is known to be race-specific and races of the pathogen can be expected to overcome the resistance within a few years. The use of polygenically inherited partial resistance, of the type expressed by Marglobe and Rutgers, would probably avoid rapid breakdowns of resistance. However, Crill, Jones and Burgis (1973; 1974) consider that this resistance is so complex that it would be unmanageable when breeding simultaneously for resistance to several diseases. Nevertheless, it is important that attempts should be made to retain, in breeding material, as many different types of resistance to each disease as possible.

Breeding for resistance to leaf mould has been only partly successful, mainly because *F. fulva* has been able quickly to develop physiologic races which can attack varieties with major genes conditioning hypersensitivity. However, the major-gene resistance of Vagabond gave an effective control of leaf mould for many years before it eventually succumbed to its own special race of *F. fulva*. In spite of the ability of this pathogen to match major resistance genes in the host with corresponding genes for virulence, there are good prospects for the

control of leaf mould by resistant varieties. Boukema and Garretsen (1975) have found 'uniform' resistance to leaf mould, which is effective against a broad spectrum of complex races of *F. fulva* in a number of tomato varieties, including many old Dutch varieties. New sources of resistance to complex races of the pathogen have also been found in progenies of crosses between the tomato variety Ace and *Lycopersicum chilense* (Yordanov, Stamova and Stoyanova, 1974). Combinations of different types of resistance should give a more durable control of leaf mould than that which has, so far, been experienced (Boukema, 1977).

Sorghum

Sorghum is the fourth most important cereal crop in the world and is grown in the subtropics and tropics under a variety of names, including Kafir corn, durra, milo, sorgo and broomcorn (Doggett, 1970). Although it is grown mainly for its grain, it is also important in some areas as a fodder crop. There are many races of cultivated sorghums, including the milo, kafir and feterita groups, each of which comprises several varieties.

Sorghums are attacked by several fungal diseases of the stem, leaf and panicle, and good resistance to most of these is available (Tarr, 1962; Doggett, 1970; Webster, 1975). For example, varieties have been developed which are resistant to milo disease (*Periconia circinata*) (Poehlman, 1959) and to rust (*Puccinia purpurea*) (Coleman and Dean, 1961). Resistant varieties also play an important part in controlling smut diseases (*Sphacelotheca* spp.) of the panicle which can cause severe crop losses in most sorghum-growing areas. Resistance to leaf blight, caused by *Exserohilum* (*Helminthosporium*) *turcicum*, is known to occur in sorghum, for example in TAM 2572, a line of *Sorghum bicolor* (Tuleen and Frederiksen, 1977).

SMUTS

Three smut diseases are of economic importance in sorghum. These are covered smut (*Sphacelotheca sorghi*), loose smut (*S. cruenta*) and head smut (*S. reiliana*). Covered and loose smuts can usually be controlled adequately by seed dressings of fungicides, and head smut, which is soil-borne, can be partly controlled by appropriate crop rotation. The cheapest and best method of control, however, is to use resistant varieties.

Covered smut is one of the most common and damaging diseases of sorghum, particularly in North America. Seedlings become infected by spores carried on the seed, and the fungus spreads systemically within the plant. Inflorescences of infected plants bear masses of black spores of *S. sorghi*, which are surrounded by a membrane, in place of the grain. The membrane ruptures, releasing spores which settle on grain of healthy plants, thus completing the infection cycle of the pathogen.

Work on breeding for resistance to covered smut in the 1920s showed that some feterita sorghums, particularly Spur Feterita, are highly resistant, and that resistance is a dominant character (Reed, 1930). Later studies by Casady (1961) showed that resistance to races 1, 2 and 3 of *S. sorghi* is controlled by three

dominant genes, designated Ss_1 , Ss_2 and Ss_3 respectively, which are carried on the same chromosome. Crosses between the susceptible variety Pink Kafir (ss_1 , ss_2 , ss_3) and Spur Feterita (Ss_1 , Ss_2 , Ss_3) suggest that resistance to covered smut is incompletely dominant.

Five physiologic races of *S. sorghi* have been distinguished by their reactions on differential varieties (Melchers, Ficke and Johnston, 1932). Several types of sorghum including kafir, sorgos, Sudan grass and broomcorn are susceptible to all five races; some selections from Spur Feterita are resistant to all. A programme of breeding for resistance to covered smut using Spur Feterita as the source of resistance was started in Kansas in 1931 and resistant varieties were successfully developed. Many of the early resistant varieties from this programme were less resistant than Spur Feterita, but varieties with good resistance to both covered smut and head smut were subsequently released (Casady, Heyne and Hansing, 1962).

Spur Feterita is also resistant to loose smut (*S. cruenta*) and head smut (*S. reiliana*), and many of the varieties derived from this sorghum are resistant to all three smut diseases. There is probably a close linkage between genes for resistance to the three pathogens (Stewart and Reyes, 1958; Casady *et al.*, 1962). Good progress has been made in producing varieties with resistance to *S. reiliana*, particularly in the USA and India, but resistance-breaking races of the pathogen are a serious threat (Al-Sohaily, Mankin and Semeniuk, 1963; Padaganur and Govindu, 1971; Frederiksen, Rosenow and Reyes, 1975). For example, race 3 of *S. reiliana* attacked previously resistant varieties in Texas during the late 1960s, becoming the dominant race there in 1974. A population of the pathogen (race 4), which can attack sorghums with resistance to race 3, was discovered in Texas in 1974 and this threatens other resistant cultivars (Frederiksen *et al.*, 1975). This race-specific resistance seems to involve hypersensitivity (Wilson and Frederiksen, 1970).

MILO DISEASE

Periconia circinata causes a very destructive root and stalk rot disease of the milo group of sorghum and milo derivatives. Infected plants are stunted with yellow and rolled leaves and, if they survive to maturity, yield little grain. The disease is soil-borne, surviving in the soil as thick-walled chlamydospores. The fungus spreads from plant to plant by air-borne brown or black conidia. Resistant varieties have been the main method of controlling milo disease, and an important attribute of new milo varieties is an acceptable level of resistance to this disease.

A few apparently healthy plants of Dwarf Yellow milo were found in a field of plants severely infected with *P. circinata* in Kansas in 1930; progenies of these plants were subsequently grown in infected soil and were found to be resistant to milo disease (Poehlman, 1959). Other resistant plants were found by growing milo varieties in infected soil, either in the field or in the glass-house, and plants that did not become badly infected were used as sources of resistance. The specific toxin from *P. circinata* has also been used in mass screening tests for resistance to milo disease (Schertz and Tai, 1969). Plants that are very susceptible to low concentrations of toxin are badly affected and can easily be distinguished from resistant plants. Many resistant varieties have been developed

and these have given a good control of milo disease. For example, in a series of field experiments in Texas, susceptible varieties yielded only 44–76 per cent of the grain yield of resistant varieties when grown in infected soil.

Some reports suggest that resistance to milo disease is partially dominant and controlled by a single gene (Coleman and Stokes, 1954) but others indicate that susceptibility is partially dominant (Doggett, 1970). This apparent contradiction may imply that more than one type of resistance is involved and more information concerning the nature and genetics of resistance to *P. circinata* is desirable. There seem to be no published reports of any serious breakdowns of resistance associated with the development of resistance-breaking races of *P. circinata*. Although resistance is controlled by major genes it has been durable, and milo disease is no longer of great economic consequence as a result of the widespread use of resistant varieties.

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The references cited in this chapter, together with those for Chapter 3, are listed in References – Part II, pages 140–166.

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BACTERIAL DISEASES

Economic Importance of Bacterial Diseases

Although, in general, bacterial diseases are much less damaging to crop plants than are those caused by fungi and viruses, there are several very important bacterial pathogens of major agricultural crops (Cramer, 1967). These include the pathogens which cause bacterial blights of rice and cotton, bacterial wilts of tobacco and tomato and fireblight of pears and apples. Bacterial pathogens also cause important soft-rot diseases in many crops, including potatoes and carrots.

Bacterial blight of cotton (*Xanthomonas malvacearum*) causes significant losses of yield in many parts of the world. For example, yield losses from this disease in Uganda have exceeded 20 per cent in fields sown with untreated seed and about 10 per cent in fields sown with seed treated with a suitable bactericide. In the USA bacterial blight is one of the most serious diseases of cotton in spite of many control measures and is estimated to reduce the annual average crop yield by more than 2 per cent. *Xanthomonas oryzae*, which has been recognized as a serious pathogen of rice in Japan for more than 70 years, causes a very damaging disease in most of the major rice-growing areas of Asia.

Pseudomonas solanacearum, which can attack a wide range of crop plants, can cause significant damage to many crops including bananas, tobacco, tomatoes and groundnuts. It is particularly damaging to tobacco in Bengal and in the USA, where it is responsible for yield losses of about 2 per cent. In South America this pathogen causes the serious 'Moko' disease of bananas which can decrease yields of fruit by more than 15 per cent.

Pseudomonas tabaci, the tobacco wildfire bacterium, is responsible for yield losses of nearly 12 per cent in the USA and a related pathogen, *P. angulata*, can severely damage the tobacco crop in parts of East Africa.

Several species of *Erwinia* cause very serious diseases in many crops, for example fireblight of pears and apples (*E. amylovora*) which is the second most important disease of these fruit crops in North America. Other species of *Erwinia* are responsible for heavy losses by causing soft-rot diseases of many crops including potatoes and peppers.

Some Characteristics of Bacteria and Bacterial Diseases

Plant pathogenic bacteria can be classified into five main genera: *Agrobacterium*, *Corynebacterium*, *Erwinia*, *Pseudomonas* and *Xanthomonas*. Most of the

bacteria causing important plant diseases are found in the last three genera. Bacterial pathogens can also be divided into three major groups according to their primary effects on host cells (Kelman and Sequeira, 1972): (1) gall-forming bacteria, which alter the pattern of growth of the host plants; (2) soft-rot bacteria, which macerate cell wall components; (3) bacteria which alter the physiology or metabolism of individual host cells. This last group contains many of the most damaging plant pathogens which cause leaf spots, blights, cankers or wilts.

Bacteria enter plants through natural openings, such as stomata or lenticels, or through wounds; pathogenic bacterial species can then multiply in the intercellular spaces or in the xylem vessels of susceptible plants. This multiplication occurs without any direct contact between the bacterial cells and the protoplasts of the host cells, and in this respect bacteria are unlike any other plant pathogens (Kelman, 1972). Saprophytic bacteria do not multiply in the intercellular spaces or xylem vessels, although these contain all the nutrients which the organisms require for their culture *in vitro*. This suggests that, in spite of the apparently 'distant' relationship between host and pathogen there is, nevertheless, a specific interaction between them.

Bacteria in the genus *Pseudomonas* are motile, Gram-negative, straight or curved rods with polar flagellae. The genus is divided into two principal groups of species on the basis of their requirements for different growth factors; a key which characterizes the various species has been prepared by Schroth and Hildebrand (1972).

The genus *Xanthomonas* comprises about 120 species, all of which are plant pathogens. Many of the distinctions between these species are of doubtful significance, however, and Lelliott (1972) recognized only five taxospecies, namely *Xanthomonas campestris*, which contains many host-differentiated pathotypes, *X. albilineans*, *X. axonopodis*, *X. ampelina* and *X. fragariae*.

Bacteria in the genus *Erwinia* are Gram-negative, peritrichously flagellated rods which usually cause soft-rot diseases of plants. The genus contains two major groups of plant pathogens: (1) the soft-rot coliform bacteria, including *Erwinia carotovora*, (2) those which like *E. amylovora*, cause wilts and necrosis (Graham, 1972).

Bacterial pathogens usually spread from plant to plant within a susceptible crop by wind or splash dispersal. A few bacterial diseases can be transmitted through the seed. For example, *Pseudomonas phaseolicola*, which causes halo blight of dwarf and runner beans, can pass directly into the developing seeds of infected plants so that the seed coat or cotyledons become contaminated with bacterial cells. *Xanthomonas malvacearum*, the bacterial blight pathogen of cotton, is also often transmitted through the seed.

Bacterial plant pathogens can also be transmitted by insects. *Erwinia amylovora* can be carried from flower to flower by honey bees and other insects that are attracted by gummy exudates, containing numerous bacterial cells, from twig cankers on infected apple or pear trees. The pathogen that causes bacterial wilt of maize can be transmitted by flea beetles in which it can overwinter: the vector can thus introduce the pathogen into healthy maize crops in the following spring.

Bacterial diseases can be controlled by cultural means, by chemicals including antibiotics, and by resistant varieties. Bacterial pathogens can survive between successive susceptible crops in infected soil or seed, in infected crop plants, or

in alternate host plants, which may include many common weeds. Bacteria are difficult to control in the soil under field conditions although, in glasshouses, soil sterilization by steam or chemicals can be very effective. Crop rotation involving non-host species can help to avoid severe attacks, and is an important part of integrated control programmes with such bacterial diseases as bacterial wilt of tobacco and bacterial blight of rice. Crop rotation is unlikely to give a complete control of any bacterial disease, however, because bacteria can survive at very low population densities in the soil, even in the absence of host plants, for many years (Goto, 1972).

Bacterial blight of cotton usually spreads by infected seeds; seed dressings with mercury or other appropriate bactericides have helped to control the disease. Antibiotics have been used successfully to control bacterial blight, but their widespread use could possibly give rise to medical and environmental problems. In addition, antibiotics and other bactericides are expensive, and can be laborious and costly to apply. Manufacturers of agrochemicals are apparently reluctant to produce and market new chemicals to control plant bacterial diseases, perhaps because of the expense involved and the rather limited market for such products. It is not surprising, therefore, that breeding for resistance to many bacterial diseases has been an important breeding objective and many resistant varieties of several crop species have been developed.

Other forms of biological control, particularly by bacteriophages which are bacteria-infecting viruses, do not seem to have been explored to the full. Bacteriophages might be very effective in protecting plants from bacterial diseases (Civerlo, 1972).

Variability of Bacteria

Reproduction of bacteria is usually asexual and involves the division of each cell into two parts. Two daughter DNA molecules are formed from the DNA molecule which constitutes the bacterial chromosome. In many bacteria there are DNA fragments in the cytoplasm in addition to the large circular chromosome. Some of these fragments may become incorporated into the main chromosome during cell division, forming new bacterial genotypes. Other variations arise from genetic recombination, which involves the transfer of part of the DNA from one bacterial cell to another. This transfer can be effected by bacteriophage viruses or during conjugation between adjacent bacterial cells. It allows blocks of genetic material to be exchanged between individuals and can be important in enabling bacteria to adjust to new environments, including resistant plants.

The most important sources of variation in bacteria are mutations; it has been calculated that in a culture of *Escherichia coli* there is, on average, one mutant cell in every 2000 cells. There are no equivalent estimates for mutants of plant pathogenic bacteria, but there is no reason to suppose that they have a lower mutation rate than *E. coli*. Bacteria can reproduce very quickly under optimal conditions and the multiplication rate of mutant types would be very rapid if they were strongly favoured by selection pressure, as for example on plants with race-specific resistance.

It is not surprising, therefore, that bacteria are so variable genetically and that new variants which can attack previously resistant varieties have sometimes

been a serious problem, particularly in the case of the bacterial leaf blight diseases of cotton and rice. Nevertheless, resistance-breaking races have not generally caused severe damage in the field in most crops (Crosse, 1975), including tobacco, tomatoes, apples and pears; this contrasts strongly with the situation regarding resistance to many fungal pathogens.

Types of Resistance

Although *Xanthomonas malvacearum* can, apparently, penetrate unwounded cuticle between the epidermal cells of cotton leaves (Nayudu, 1963), most pathogenic bacteria can enter their host plants only through natural openings or wounds. It is unlikely, therefore, that physical barriers are an important factor in host-plant resistance. Nevertheless, the concentration, size and morphology of stomata or lenticels and their distribution on the surface of the host plant, which are genetically controlled characteristics, may influence the susceptibility of particular host genotypes to bacterial infection.

Differences between resistant and susceptible plants do not usually become evident until bacteria have entered the intercellular spaces or xylem vessels of the host. The resistance which can then be expressed is of two main types: (1) resistance caused by factors which existed before inoculation; (2) induced resistance following a response of the host plant to inoculation.

Pre-existing resistance factors can include several different kinds of mechanisms. For example, the intercellular fluids of the host may be unsuitable for the rapid multiplication of a particular pathogen because of unsatisfactory buffering capacity or osmotic potential. A lack of suitable nutrients in these fluids may inhibit bacterial growth. Nayudu (1963) reported that high concentrations of serine, a free amino acid, inhibit the multiplication of *Xanthomonas malvacearum*, but it seems that the intercellular fluids of resistant varieties of cotton do not necessarily contain higher concentrations of serine than those of susceptible varieties. Kelman and Sequeira (1972) consider that such factors are unlikely to explain why closely related strains of a bacterial pathogen can attack one variety of a crop plant but not another.

Several compounds that are known to inhibit the multiplication of bacterial pathogens *in vitro* have been detected in healthy plants. These include many phenolic compounds (Kelman and Sequeira, 1972) and an unidentified inhibitor in maize plants which is more effective against those species of *Erwinia* that attack maize than against those that do not (Hartman, Kelman and Upper, 1972). Tomato varieties which are resistant to *Pseudomonas solanacearum* contain higher concentrations of the glycoalkaloid tomatine, an effective inhibitor of bacterial growth *in vitro*, than do susceptible varieties. High concentrations of tomatine are also associated with resistance of tomatoes to many other pests and diseases (Zhuchenko, Balashova and Andryushchenko, 1974). Some of these pre-existing resistance factors may, therefore, be implicated in generalized resistance to bacterial diseases and are worthy of further study.

Kelman (1972) has pointed out that, although many plant-pathogenic bacteria release in the host one or more physiologically active compounds, including proteolytic, cellulolytic and pectolytic enzymes and toxins, none of these has been shown conclusively to induce a specific defence reaction in a host plant. Incompatible reactions between particular host and bacterial genotypes

usually involve hypersensitivity. In a hypersensitive reaction, bacterial cells passively penetrate into a substomatal cavity of the host and start to multiply: host cells adjacent to the bacterial cells die as a direct response to infection, and the bacterial cells subsequently also die. This confines the infection, so that the host plant as a whole remains almost free of symptoms (Klement, 1972). Hypersensitivity may produce macroscopic changes in the host plant, for example chlorotic or necrotic local lesions which are visible to the naked eye, or lesions involving only a few dead or damaged cells, which may be visible only under a fluorescence microscope after appropriate staining of the tissues. The mechanisms which are involved in hypersensitivity to bacterial pathogens are not fully understood but Klement (1972) showed that three main stages could be distinguished in hypersensitivity of tobacco to *Pseudomonas phaseolicola*: (1) an **induction period** of 3–4 hours after inoculation, which is the minimum time for this pathogen to initiate a hypersensitive response, which subsequently is irreversible; (2) a **latent period** of 4–5 hours between the end of the induction period and the onset of tissue collapse; (3) **tissue collapse**, which involves the disorganization and malfunction of the cell membrane. This leads, directly or indirectly, to the death of the affected cells. Phenolic compounds and other toxins are released by damaged host cells, which apparently kill both themselves and neighbouring bacterial cells.

Bacterial pathogens are thought to contain three factors which elicit or avoid the hypersensitive response of a host plant (Kelman, 1972). The first is concerned with the induction of hypersensitivity, and the second with prevention of the development of a hypersensitive reaction. The third factor prevents the release of the first two factors or interferes with their effects on the host; this factor, therefore, determines host specificity. It is probable that specific proteins in bacterial cells are involved in the induction and prevention of hypersensitivity. Certain antigens, presumably proteins, are shared by *Xanthomonas malvacearum* and cotton (Devay, Schnathorst and Foda, 1967) and these common antigens may determine the host specificity of bacterial pathogens.

Many soft-rot bacteria do not elicit a hypersensitive response in their host plants, and resistance mechanisms other than hypersensitivity have to be envisaged. Resistant plants may, in some way, be able to inhibit the activity of enzymes that are responsible for macerating and degrading host cell constituents. The precise nature of resistance to bacteria is not understood; several different resistance mechanisms are probably involved.

Hypersensitivity, as with diseases caused by fungi, seems very often to be associated with race specificity and 'breakdown' of resistance; other kinds of resistance may be more durable. More information is urgently needed about the role of pre-existing defence mechanisms and, in particular, about the compounds resembling interferons and antibodies that are produced by many plants in response to inoculation with certain plant pathogenic bacteria (Kelman, 1972). Such compounds may play a key part in resistance to bacterial pathogens.

Until more information is available about the nature of resistance, plant breeders must continue to rely on empirical methods of selecting for resistance to bacteria. In the meantime, it may be helpful to distinguish between three main types of resistance, namely disease escape, restriction of area of host plant infected, and disease tolerance. Disease escape is manifested by fewer infections, either on whole plants or on individual organs such as leaves or roots. The restriction of infection will result from the effects of hypersensitivity or of any

resistance mechanism which affects the translocation or multiplication of bacteria. Tolerance, which seems to have been exploited less by breeders selecting for resistance to bacteria than by those selecting for resistance to viruses, fungi and animal pests, is expressed as decreased damage to a host plant even when it supports large populations of bacteria. Methods which can be used to test for different types of resistance to bacteria are described in a subsequent section of this chapter (see page 176).

Sources of Resistance

Varieties and wild species have both been widely used as sources of resistance to bacterial diseases. For example, more than 1000 different types of cottons, including varieties of *Gossypium hirsutum* and *G. barbadense* and wild plants of related species, were screened to find sources of resistance to *Xanthomonas malvacearum* (see page 179). Several sources of resistance were identified and these have been used in the production of resistant varieties. Cultivated varieties and wild species have also been used as sources of resistance to *Pseudomonas solanacearum* (see page 190) and *Corynebacterium michiganense* (see page 193) in tomato and to *Erwinia amylovora* in pears and apples (see page 195). On the other hand, the main sources of resistance to *Pseudomonas solanacearum* in tobacco and to *Xanthomonas oryzae* in rice have come from cultivated varieties rather than wild relatives. However, no good sources of resistance to *Pseudomonas tabaci* seem to have been found in cultivated tobaccos, whereas resistance derived from *Nicotiana longiflora* has been transferred successfully to *N. tabacum* with the result that several wildfire-resistant tobacco varieties have been bred, including several Burley varieties (see page 185).

It is sometimes difficult to transfer resistance genes from wild species to cultivated species without also transferring undesirable characteristics. Fortunately, such problems do not seem to have been important in breeding for resistance to most bacterial diseases. However, it has been difficult to develop tomato varieties which are resistant to bacterial wilt, and which yield fruit of high quality, with resistance derived either from wild species or from cultivated varieties.

Resistance-breaking variants of bacterial plant pathogens do not seem to have been more common with any one type of resistance source than with any other. It can, therefore, be concluded that there are no particular disadvantages in the use of wild species as sources of resistance. It should be remembered, however, that resistant varieties of an acceptable standard of yield and quality are likely to be developed more quickly and more easily from resistance sources that are already in the genetic background of a cultivated variety.

Inheritance of Resistance

Although major genes have been reported to control resistance to many bacterial pathogens, the genetics of resistance to bacterial diseases is often controversial. For example, the expression of resistance to *Xanthomonas malvacearum* in cotton can vary greatly in different parts of the world; different workers have, therefore, reached conflicting conclusions about the genetic

control of resistance. Arnold and Brown (1968) suggested that these discrepancies were attributable to strong genotype—environment interactions involving polygenic systems, in both host plant and pathogen. Studies of the inheritance of resistance to *Xanthomonas oryzae* in rice have given similar conflicting results. Some workers have reported that resistance to *X. oryzae* is monogenically controlled, and others have indicated that many resistance genes are involved. Resistance has also been reported by different workers as being a dominant or a recessive character. These results suggest that the genetics of resistance depends not only on the genotypes of the host and pathogen concerned, but also on the environmental conditions in which these genotypes are grown.

The control of resistance to *Pseudomonas solanacearum* has been reported to be dominant by some workers and recessive by others and, here again, the expression of resistance can be greatly influenced by environmental changes. Resistance to *Pseudomonas tabaci* in tobacco segregated in Mendelian ratios in crosses between certain resistant and susceptible varieties but not in others, suggesting that expression of resistance to this pathogen is also labile.

It seems, therefore, that the expression of resistance to many bacterial diseases can be influenced to a greater extent by changes in environmental conditions than is the case with most types of resistance to fungal and virus diseases. This not only complicates studies concerning the genetic control of resistance and selection for resistance, but it can also determine the effectiveness of disease control by resistant varieties in different environments.

Methods of Selection and Breeding

Most of the early attempts to select for resistance to bacterial diseases were carried out where natural disease epidemics could be expected to occur. For example, this method was used for many years to select and test for resistance to *Xanthomonas oryzae* in rice in Japan, although artificial inoculations were used in later tests. As might be expected, tests carried out under natural conditions generally reflect the effectiveness of field resistance more accurately than glasshouse or laboratory tests. There are many other advantages of using natural disease epidemics, including saving time and labour in preparing and applying inoculum of the causal pathogen. It is usually possible to obtain additional information from field tests, regarding important host plant characteristics other than resistance to disease, such as yield and quality of produce. Natural field epidemics may not occur every year and it is often necessary to encourage epidemics by sowing infected seed, by planting the test material in infected soil or by interplanting rows of susceptible varieties between rows of the varieties to be tested for disease resistance.

Although there are many advantages in carrying out initial large-scale preliminary selection tests, and perhaps final evaluation tests of resistant varieties, under conditions of natural infection, it is often necessary to inoculate test plants artificially to obtain a more uniform disease epidemic than would occur naturally. This uniformity can greatly facilitate comparisons between different breeding lines and genotypes. It has, therefore, been necessary to develop suitable inoculation methods for use in selection tests in the field and the glasshouse.

Unlike viruses and obligate fungal pathogens, bacteria can usually be cultured *in vitro* without difficulty. For example, *Erwinia* soft-rot bacteria and *Pseudomonas solanacearum* can be cultured on a modified Drigalski's medium (Goto, 1972). Although most culture media are not selective for any particular species of bacteria, a selective medium for *Erwinia amylovora* has been developed by Miller and Scroth (1970). Such media can be very useful in preparing inocula for large-scale screening tests because the dangers of contamination with other species of micro-organism are greatly reduced. Cultures of most bacterial pathogens can be grown in nutrient broth in a shaker bath, or on nutrient agar slopes. Alternatively, inoculum can be prepared by macerating the tissues of infected plants in water or in an appropriate buffer solution.

Many different methods have been used to inoculate plants with bacteria in the field or in the glasshouse. One of the most widely used techniques has involved spraying the test plants with a suspension of bacteria in water, using an atomizer. This method has been used in breeding for resistance to the bacterial blight diseases of cotton (*see* page 181) and rice (*see* page 183). High-pressure sprays give the best results because bacterial cells are then injected into the stomata. Water-congested leaves are particularly susceptible to infection with bacterial pathogens and high-pressure sprays encourage a water-soaked condition of the leaves.

On a smaller scale, suspensions of bacterial cells can be injected into the intercellular air spaces of a leaf. A syringe can be used with either a hypodermic needle (Kennedy, 1969) or with a soft rubber bung without a needle (Hagborg, 1970). In the latter case, the rubber bung is pressed gently against the surface of the host plant and the plunger is slowly depressed so that the bacterial suspension passes through a hole in the bung and into the leaf through the stomata. It is, of course, necessary to ensure that at least some of the stomata are open before starting this procedure. A finger on the reverse side of the leaf or stem provides a suitable support so that an appropriate amount of pressure to inject the suspension into the tissues can be applied with the minimum amount of damage to the plant.

Needles contaminated with bacteria have been used extensively to inoculate plants in breeding programmes. In the simplest forms of needle inoculation, a single needle is dipped into a suspension of bacterial cells and is then used to prick the surface of the plant gently. Multineedle inoculation techniques, using bundles of 50 or 100 fine needles, have been devised so that many simultaneous inoculations can be made over a very small area (Isaka, 1970).

Seedlings can often be inoculated with bacteria, before they are transplanted into pots or into the field, by immersing them in a bacterial suspension. This method has the advantage that the host-plant tissues become water-soaked, thus predisposing them to disease.

The most appropriate type of inoculation method is usually that which is most convenient under the circumstances. The seedling-immersion technique is very suitable for seedling tests in the glasshouse or where seedlings are to be transplanted into the field. Spraying inoculum on to test plants by means of a high-pressure spray is particularly appropriate where large numbers of plants have to be inoculated simultaneously. It is reassuring that similar results have usually been obtained from different inoculation methods, for example in screening for resistance to *Xanthomonas oryzae* in rice.

Methods of assessing the results of infection, whether natural or artificial,

are very similar to those used for fungal and virus diseases. In assessing the disease escape tendencies of breeding material the percentages of disease-free plants after inoculation can be compared or the numbers of individual infections, for example necrotic or chlorotic lesions, can be counted on each plant. Differences in the spread of the pathogen and of symptoms within different host plants can be compared in many ways, for example by measuring the size of lesions or by assessing the proportion of the surface of the host plant which is diseased. Tolerance is best measured by comparing yields of breeding lines, both when they are healthy and when they are infected; the lines which show the smallest differences between the yields of healthy and infected plants are the most tolerant of disease. Wherever possible, those plants which are selected as being resistant to disease should be examined for the presence of desirable or undesirable characteristics. Only those resistant plants with the best combination of desirable characteristics should be selected for use in the breeding of a resistant variety.

References

The references cited in this chapter, together with those for Chapters 6, 7, 8, and 9 are listed in References — Part III, pages 267–290.

SOME EXAMPLES OF BREEDING FOR RESISTANCE TO BACTERIAL DISEASES

Cotton

BACTERIAL BLIGHT

Bacterial blight of cotton, also commonly referred to as angular leaf-spot, blackarm or boll rot, is caused by *Xanthomonas malvacearum* and occurs in all the major cotton-growing areas of the world, often causing very great losses of yield. Spotting of the leaves is often the most obvious symptom but stem infection can cause more serious damage, and infected bolls may be killed or blackened. Yield losses of more than 50 per cent have been recorded in susceptible varieties in severe field epidemics. The disease is usually spread by contaminated seed; disinfection of the seed by mercury seed dressings, and appropriate cultural practices, can give a partial control of bacterial blight. However, resistant varieties have played an important part in controlling this disease in many parts of the world.

Knight (1946) working in the Sudan carried out very important pioneering work on breeding for resistance to *X. malvacearum*. He developed a simple field screening technique in which young cotton plants were inoculated by spraying the undersurface of the leaves with a dilute suspension of bacterial cells. The severity of bacterial blight symptoms was assessed visually after two to three weeks using a grading system of 0 for absence of symptoms to 12 for complete susceptibility. More than 1000 different types of cotton, including several wild species of *Gossypium*, were screened in this way and many sources of resistance were discovered. Knight and Hutchinson (1950) concluded from these and other studies that bacterial blight had probably originated in the Indian subcontinent because so many of the resistant types had come from that area. A number of major resistance genes in different types of cotton were identified and some of these were exploited in the development of resistant varieties of *G. barbadense* (Egyptian cotton) for use under irrigation in the Sudan. The breeding programmes carried out by Knight and others in the Sudan were very successful in achieving a high level of control of the disease (Innes, 1970) and their successes undoubtedly stimulated breeding work in other cotton-growing areas. The resistance genes that were used in the Sudan included B_2 (a dominant gene from a *G. hirsutum* variety, Uganda B31); B_3

(a partially dominant gene from *G. hirsutum* var. *punctatum*) and B_6 (a 'modifier' gene from *G. arboreum* which, in combination with B_2 or B_2B_3 , confers near-immunity; B_4 , a partially dominant gene from *G. arboreum*; and B_7 , a partially dominant gene from another *G. hirsutum* variety, Stoneville 20. Several other major genes for resistance have since been discovered and certain combinations of B genes, including B_2B_9K , B_2B_6 and $B_2B_3B_6$ have been used in the development of many resistant varieties of *G. barbadense* and *G. hirsutum* (Innes, 1974). For example, Barakat, a variety of Egyptian cotton (*G. barbadense*) which is homozygous for B_2B_6 , has been grown on a large scale in the Sudan (Siddig, 1973).

Although Knight's B genes have been very widely used in many parts of the world, including many African countries and the USA, several workers have failed to obtain the expected Mendelian ratios when using the genes in breeding programmes. Arnold (1963) showed that resistance to *X. malvacearum* is considerably more complex than had hitherto been thought, the expression of resistance being greatly influenced by changes in environmental conditions. This complexity of resistance was confirmed by Innes (1970) who reported that different lines of cotton carrying the same major resistance genes can express resistance ranging from near-immunity to full susceptibility. Arnold and Brown (1968) consider that the relationship between *X. malvacearum* and its host is a function of the interaction of environmental conditions and two polygenic systems, that of the host and that of the pathogen, rather than the effects of a series of major resistance genes.

El-Zik (1968) found that lines of *G. hirsutum* cotton carrying the genes B_4 , B_2B_3 , B_2B_7 or $B_2B_3B_7$ were highly resistant to all known American races of *X. malvacearum*. Diallel analyses showed that the observed frequency distribution patterns of resistance were typical of those expected for quantitatively inherited characters, and there was some evidence of transgressive segregation. El-Zik concluded that a major part of the total expression of resistance was attributable either to additive gene effects or to an interaction of additive or dominance effects. Similar conclusions were reached in Africa by Innes and Brown (1968) and by Innes, Brown and Walker (1974), who carried out experiments at different sites in East Africa with half-diallel sets of crosses between inbred cottons. One line, 101-102B, showed outstanding resistance to bacterial blight; this resistance is controlled by a complex of polygenes and two major resistance genes, B_2 and B_3 . Owen (1967) studied the genetics of resistance to *X. malvacearum* in five American Upland cottons and found that several resistance genes were involved in one variety, two genes in another and only one gene in the remaining two varieties. In all these varieties, resistance was incompletely dominant and it appeared that additional genes which imparted greater resistance were also present. Davis, Yang and Chew (1974) reported that several genes condition the resistance of resistant Acala strains of cotton.

In spite of the complexity of the genetics of resistance to bacterial blight, considerable progress has been made in producing resistant cotton varieties in many parts of the world, including East Africa (Innes, 1974; Innes *et al.*, 1974; Ebbels, 1976), the USA (e.g. Hunter, 1960) and South America (Cia *et al.*, 1975). On the other hand, most *G. hirsutum* varieties are too susceptible to be grown in many parts of India because of the presence of *X. malvacearum* races which can attack them (Verma and Singh, 1974). However, varieties of local cotton (*G. arboreum*) have not been badly attacked by bacterial blight in India

and both *G. arboreum* and *G. herbaceum* have shown a very high level of resistance in field experiments (Jakkanwar and Bhagwat, 1971; Nagarkoti *et al.*, 1974).

Testing and selecting for resistance to bacterial blight has usually been carried out in the field either in natural epidemics of the disease or after artificial inoculation. Leaf inoculation usually involves spraying the undersurface of leaves with a suspension of bacteria obtained by soaking infected leaves in water (Knight, 1946; Innes, 1974). Stems and bolls can be inoculated by pricking them with inoculum-charged needles (Wickens, 1953). Last (1959) and Gunn (1962) developed techniques for inoculating seedlings and these have facilitated the screening of large numbers of young plants for resistance to bacterial blight. However, seedling tests cannot completely replace field tests with adult plants because the results of seedling and adult-plant tests do not always coincide. Furthermore, several workers including Wickens (1953) have reported that there is not always a close association between the resistance of leaves, stems and bolls in some genotypes; they have suggested that the susceptibility of these different organs should be considered separately in breeding programmes. However, Lagi re (1960) found positive correlations between the resistance shown by leaves, stems and bolls. Arnold (1963) also considered that leaf, stem and boll resistance is under the control of the same genetic system, but he pointed out that interactions between genotype and environment might create the misleading impression that different parts of the plant were under different genetic control. Innes (1970) found a general positive correlation between stem and leaf resistance in most of the genotypes that he tested; there is usually also a close association between field resistance in natural epidemics and resistance of the leaf to artificial inoculation (Innes, 1974).

Gunn (1961) reported that, over a period of thirteen years in the Sudan, there had been a significant loss of resistance to *X. malvacearum* in several breeding stocks, including BAR 14/25 and BAR 4/15, both of which carry the B_2B_3 resistance genes. He suggested that the most likely explanation for this apparent loss of resistance was a change in the pathogenicity of *X. malvacearum* during this period. Crosse (1963) found populations of *X. malvacearum* in Tanganyika which were able to overcome the resistance of hybrids derived from the variety Albar, which had previously been resistant. Arnold and Brown (1968) concluded that there is a continuous variation in virulence, on different cotton breeding lines, in isolates of *X. malvacearum* from East Africa and that there is no clear-cut distinction between physiologic races of the pathogen. However, in the USA, Brinkerhoff (1963) identified twelve distinct races from different parts of the world using a set of differential varieties; he found that some races could multiply readily in Acala and *G. barbadense* genotypes possessing major resistance genes, provided that they were without minor or modifying resistance genes. He concluded that complexes of minor resistance genes had evolved under steady selection pressure of bacterial blight infection over a period of more than a hundred years, and that these minor genes enhance the control conferred by major genes.

Several more races of *X. malvacearum* have been identified in the USA since Brinkerhoff's original work (Hunter, Brinkerhoff and Bird, 1968; Brinkerhoff, 1970) and two new races have been found in India (Verma and Singh, 1970). At least 17 races of the pathogen are now known to occur.

The considerable genetic variability of *X. malvacearum* is emphasized by

work carried out by Schnathorst (1970). He inoculated single-cell cultures of race 1 of *X. malvacearum* on to Stoneville 20, a resistant variety carrying the B_7 gene, and found that cells of race 2 could be isolated from the inoculated leaves after 56 days. No spontaneous change from race 1 to race 2 had occurred in Schnathorst's cultures over a period of more than ten years and he concluded from this that the specificity of *X. malvacearum* had been changed by one passage of an avirulent race through Stoneville 20. These results indicate that the resistance of varieties with only a single resistance gene might be very quickly overcome by new virulent races arising from spontaneous mutations. Similar experiments should be carried out under sterile conditions with a range of resistant and susceptible genotypes and several races of *X. malvacearum*.

There is very little published information about the nature of resistance to bacterial blight of cotton or about the resistance mechanisms involved. The types of resistance involved merit more intensive study so that some of the doubts surrounding the genetics of resistance and the relationships between the resistance of different plant organs can be resolved. Phytoalexins may be involved in resistance to bacterial leaf blight (Hopper *et al.*, 1975) but there is no evidence that phenolic compounds are implicated (Jalali, Singh and Grover, 1976). The degree of resistance to bacterial blight expressed by different cotton genotypes may be associated with the concentrations of certain free amino acids in the stems and leaves; Lipke (1968) found such an association in F_2

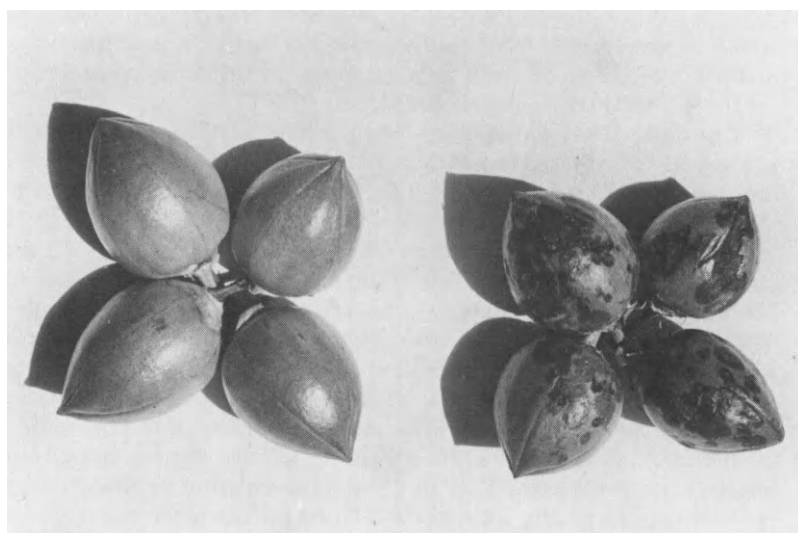


Figure 6.1 Cotton varieties differ greatly in resistance to bacterial blight caused by *Xanthomonas malvacearum*. The photograph shows inoculated bolls from a resistant (left) and a susceptible (right) variety. (By courtesy of Dr L. Innes, National Vegetable Research Station, Wellesbourne, Warwick)

and F_3 generations of *G. hirsutum* derived from crosses involving the B_4 resistance gene and minor resistance genes; there was no evidence, however, that the concentration of any of these amino acids is under the direct control of the B_4 gene. Vohra and Chand (1971) found that two susceptible varieties had higher contents of total free amino acids than two resistant varieties. No threonine,

DL-alanine or arginine was detected in the most resistant variety, although these amino acids were present in the susceptible varieties; cystine and lysine were present in the most resistant variety, but not in the susceptible varieties. However, there is no direct evidence of a causal relationship between the concentration of any free amino acid and resistance to bacterial blight. Nevertheless, even an indirect association between amino acid concentration and resistance might be a useful indicator of the level of resistance, when screening breeding material for resistance to *X. malvacearum*.

It is evident that resistant varieties of cotton have already made a very considerable contribution to the control of bacterial blight in many parts of the world (see Figure 6.1). However, the expression of resistance is very labile and is also very sensitive to changing environmental conditions; the development of more virulent strains of the pathogen, capable of attacking previously resistant varieties, must be expected. It is unlikely, therefore, that other forms of control, particularly chemical seed dressings, will become unnecessary. A combination of chemical seed treatments and resistant varieties will probably give an adequate and long-lasting control of this potentially very serious disease.

Rice

BACTERIAL BLIGHT

Bacterial blight (*Xanthomonas oryzae*), or bacterial leaf blight, has been recognized in Japan for more than 70 years as a serious disease of rice. It is also present in most rice-growing areas of Asia, but is apparently absent from Europe, Australia and most of Africa, (Ou, 1972a). The disease has only recently been reported in America (Lozano, 1977; Ou, 1977).

Symptoms of the disease develop mainly on the leaf blades and leaf sheaths. Water-soaked lesions appear at the margins of infected leaves near the tip of the lamina; in susceptible plants the lesions gradually enlarge along the veins and become yellow. In severe infections lesions eventually cover the whole leaf blade and turn white or grey. Entire leaves or whole plants may wither when infected with virulent isolates of the pathogen and when climatic conditions are ideal for spread and multiplication of *X. oryzae*. Grain yields of susceptible varieties are often decreased by 20–30 per cent by the disease in wet seasons (Exconde, Opina and Phanomsawarn, 1973) and yield losses of nearly 50 per cent have occasionally been reported. Full accounts of the symptomatology, epidemiology and control of bacterial blight have been published by Ou (1972a, b) and Mizukami and Wakimoto (1969). Although satisfactory control can often be achieved by application of antibiotics or other bactericides, or by appropriate cultural practices, resistant varieties remain the most promising method of control. Many resistant varieties have been developed, particularly in Japan, and these have undoubtedly helped to decrease yield losses caused by the disease (Ou and Jennings, 1969).

The earliest selections for resistance were made in naturally infected fields in Japan, although artificial inoculations with suspensions of bacteria were used in later selection tests. Commonly used methods of inoculation include spraying with a bacterial suspension, pricking with a needle contaminated with bacteria or immersing seedlings in a bacterial suspension, all of which are reported to give

comparable results (Reitsuma and Shure, 1950). Another method involves clipping the tips of rice leaves with scissors, the blades of which have been dipped in a suspension of bacterial cells (Kauffman *et al.*, 1973). Padmanabhan (1969) conducted mass screening tests in the field, with seedlings sown in beds that were flooded with a bacterial suspension four weeks later; plants were subsequently reinoculated by clipping leaf tips and spraying with inoculum so that the susceptibility of different lines to *X. oryzae* could be assessed at all growth stages throughout the season.

Some workers have reported that resistance to bacterial leaf blight can be detected effectively at the seedling stage (e.g. Washio, Kariya and Toriyama, 1966), and that there is a good correlation between the expression of resistance at the seedling and flag leaf stages (Ou, Nuque and Silva, 1971a). Conversely, Rao and Reddy (1975) did not find a good correlation between the seedling and adult-plant reactions to leaf blight in a number of lines of rice; they concluded that screening for resistance should be carried out with adult plants at about the heading stage when the disease is usually most pronounced. Horino and Ezuka (1973) found differences between the expression of resistance at the seedling and adult plant stages in the variety Wase Aikoku 3; the resistance gene in this variety does not apparently operate fully at the seedling stage. It seems, therefore, that although seedling tests can provide useful guidance in selecting for resistance to bacterial blight, field tests with adult plants are likely to give more reliable results.

The first resistant varieties to be released in the early 1920s in Japan included Shigasekitori II, Sugaippon, Aka-shinriki, Kanto 35 and Syobei (Mizukami and Wakimoto, 1969; Ou, 1972a, b), and several resistant varieties have been developed from them. For example, the varieties Hoyoku, Kokumasari and Ooyodo were derived from Shigasekitori II, Nihonbare and Sachikase were derived from Syobei, and Norin 27, Asakaze and Nishika were derived from Kanto 35. Although all these, and other Japanese resistant varieties, can be attacked to varying degrees by different isolates of the pathogen, they have played an important part in reducing damage resulting from bacterial blight in Japan.

Since the mid-1960s a large programme of breeding for resistance to *X. oryzae* has been carried out at the International Rice Research Institute (IRRI) in the Philippines (see *Annual Reports of International Rice Research Institute 1968-76*). Ou *et al.* (1971a) tested nearly 9000 rice varieties for resistance to bacterial blight but detected no immune types, although many partially resistant varieties were identified. Varieties that were resistant in field tests with one isolate of *X. oryzae* were retested as seedlings against several isolates of the pathogen collected in the Philippines. Varieties that had been classified as resistant in Japan were often found to be susceptible against these isolates, indicating that the resistance of many Japanese varieties is race-specific. Many varieties, including Sigadis, Dawn, TKM6, CI 9210 and several *japonica* varieties from Taiwan, have been used as sources of resistance to bacterial blight. However, none of these varieties was resistant to all the isolates of *X. oryzae* that were used in the tests. Nevertheless, several varieties with broadly based resistance to leaf blight have been developed in the Philippines.

Breeding for resistance has also been carried out in India. Srivastava *et al.*, (1967), working in New Delhi, tested 128 varieties from many parts of the world for resistance to five virulent isolates of *X. oryzae* in induced field

epidemics; only 13 of these varieties were classified as resistant to bacterial blight, with a further 11 moderately resistant. Most of the resistant varieties, including Tainan 3 and Taichung 65, were *japonica* types but two *indica* lines from IRRI were also identified. Chattopadhyay, Mukherjee and Biswas (1968) reported that two introduced types, PI 215936 and IR 30, and Muhurikhasa (a local West Bengal variety) showed resistance to *X. oryzae* under Indian conditions. Devadath and Padmanabhan (1969) found that differential reactions between 20 rice varieties and nine isolates of *X. oryzae* were more pronounced at the seedling stage than at the flag-leaf stage and that there were sometimes different interactions between the same varieties and isolates at different growth stages. These complex interactions involving genotype, growth stage and pathogen isolate may help to explain some of the conflicting reports concerning the inheritance of resistance to bacterial blight.

In 1957 a previously resistant variety, Asakaze, was severely attacked by a virulent isolate of *X. oryzae* in Japan (Kuhara *et al.*, 1965), and this stimulated large-scale work on variants of the pathogen. Sakaguchi, Suwa and Murata (1968) classified Japanese isolates of *X. oryzae* into three main groups according to the varieties which they could attack. About one-quarter of the varieties tested, mostly from North and South America, South Asia and China, were resistant to Group I isolates. About 8 per cent of the varieties, mainly those from South Asia, were resistant to Group II isolates. Line TKM6 from the Philippines and Nigeria 5 from Sri Lanka were resistant to Group III isolates, as were some lines of several other species of *Oryza* including *O. minuta*, *O. eichingeri*, *O. officinalis* and *O. granulata*. More recently, Ezuka and Horino (1974) have tested 149 rice cultivars for resistance to three 'pathotypes' of *X. oryzae* collected from different parts of Japan. Populations of these pathotypes were divided into 11 subgroups according to their virulence to individual varieties of the Wase Aikoku group. These results confirmed that the resistance in many Japanese varieties is highly race-specific. Nevertheless, some varieties, including 'Lead' rice, from Burma, TKM6 and Nigeria 5, have shown good resistance to all three Groups of isolates in recent tests and are promising sources of broad-spectrum resistance to bacterial blight (Sakaguchi, 1977). 'Lead' rice might be a particularly useful source because it is resistant also to the major races of the blast disease fungus.

Ou *et al.* (1971b) found major differences in virulence between isolates of *X. oryzae* collected in the Philippines, but could find no evidence at that time of distinct physiologic races that were adapted to specific rice varieties. Later work in the Philippines, however, produced clear evidence of race specificity between rice varieties and *X. oryzae* isolates (Reddy and Ou, 1976). Mew and Vera Cruz (1977) have used a set of four rice varieties (IR8, IR20, IR1545-339 and DV85) to differentiate strains of the pathogen in the Philippines. Many tall and semi-dwarf varieties have expressed resistance to a broad spectrum of pathogen isolates that have so far been identified. However, a virulent strain of *X. oryzae* that can attack not only IR20, which is grown in many Asian countries and is generally considered to be a resistant variety, but also related varieties, has recently been identified in South Asia (see *International Rice Research Institute Annual Report for 1973*). This shows that resistance-breaking variants of *X. oryzae* are a serious threat to resistant rice varieties which are extensively grown over a wide area.

The inheritance of resistance to bacterial leaf blight in rice has been very

intensively studied. For example, Washio *et al.* (1966) tested 44 rice varieties against 11 isolates of *X. oryzae* collected from different parts of Japan, and classified the varieties into four groups, I (resistant) to IV (susceptible), and the pathogen isolates into three groups, A, B and C. Resistance to Group A isolates of *X. oryzae* in the variety Kidama (Group III) is apparently controlled by two complementary dominant genes, X_1 and X_2 . However, resistance to lesion development is controlled by polygenes in some varieties with Group A isolates of the pathogen, but by major genes in other varieties with Group B isolates.

Sakaguchi (1967) studied the genetics of resistance in the F_2 generations of crosses between resistant and susceptible varieties, and concluded that resistance to isolates X17 and X14 of *X. oryzae* in the Rantaj-emas group of varieties is controlled by two dominant genes, Xa_1 and Xa_2 , both of which are on chromosome 11. Resistance to 'isolate 72' of *X. oryzae* is conditioned by one pair of alleles, with resistance being incompletely dominant (Heu, Chang and Beachell, 1968). The resistance of several varieties is governed by single dominant genes, for example BJ1 (Jayaraj, Seshu and Shastry, 1972; Murty and Khush, 1972; Moses, Rao and Siddig, 1974) and Kogyoku (Ezuka *et al.*, 1975). However, the resistance of other varieties, for example Tatep and Malakit Sungsong, is controlled by two closely linked genes and two complementary genes, respectively. On the other hand, resistance in BZ 192 is conditioned by two recessive genes (Murty and Khush, 1972). The inheritance of resistance to bacterial blight of rice is therefore very complex; it may be recessive or dominant, and conditioned by major genes or polygenes, depending on the particular rice genotypes and pathogen isolates that are involved (Singh and Nanda, 1975; Mohiuddin and Kauffman, 1975; Olufowote, Khush and Kauffman, 1977). The situation is further complicated because the expression of resistance is subject to maternal influence (Nayak, Ratho and Mishra, 1975; Ratho *et al.*, 1976), presumably because of the presence of extrachromosomal genes.

Very little is known about the nature of resistance to bacterial blight in rice. Mizukami (1961) found that there is an inverse correlation between resistance to bacterial blight and hydathode frequency on the leaves. Kiryu and Mizuta (1955) suggested that plants with short, narrow leaves are the most resistant, but later work has indicated that differences in the morphology of the leaves are not of overriding importance in resistance (e.g. Mizukami and Wakimoto, 1969).

Uehara (1960) observed that a non-specific antibiotic substance with phytoalexin-like properties is produced by rice plants in response to inoculation with *X. oryzae*; this compound is similar to, or identical with, that produced in response to inoculation with *Piricularia oryzae*, the blast fungus (*see* page 112). Purushothaman (1975) reported that the concentration of phenolic compounds increased more in resistant varieties such as TKM6 than in susceptible varieties, after inoculation with *X. oryzae*; two compounds that reacted with diazotized sulphanilic acid were present in TKM6 but not in the susceptible varieties tested. However, the importance of phytoalexins and other phenolic compounds in resistance to bacterial leaf blight is uncertain.

Several attempts have been made to relate the concentration of sugars and free amino acids in the leaves with their resistance to bacterial blight. For example, Fang *et al.* (1963) found that susceptible varieties have higher contents of certain free amino acids and lower contents of some polyphenols and reducing sugars than resistant plants. Heavy applications of nitrogenous

fertilizers increased both susceptibility to bacterial blight and the concentration of free amino acids and amides in the leaves; plants deprived of nitrogen were resistant to *X. oryzae* and contained high concentrations of sugars and polyphenols. Previous work by Mizukami and Murayama (1960) had also suggested that high concentrations of certain free amino acids are associated with susceptibility to bacterial blight, and it has been confirmed that the development of the disease is often closely related to the sugar content of the leaves (International Rice Research Institute, 1970). These results suggest that simple chemical tests for estimating the content of free amino acids and sugars might be useful in selecting and testing for resistance to bacterial blight.

Breeding for resistance to bacterial leaf blight in rice has been complicated by the great genetic variability of *X. oryzae* (Ou *et al.*, 1971b). It seems that all the resistant varieties that have been developed so far are susceptible to at least one of the known isolates of *X. oryzae*, and no good sources of non-race-specific or durable resistance to bacterial leaf blight have yet been found (Kauffman and Rao, 1972). Although resistant varieties are widely cultivated in many parts of Asia, even these varieties can be severely damaged by virulent isolates of *X. oryzae*. It is not possible, therefore, to rely solely on resistant varieties at present for an adequate control of bacterial leaf blight, and an integrated control programme, involving resistant varieties, chemical methods and cultural measures, is widely practised.

Certain combinations of major resistance genes in individual varieties, or the development of multiline varieties, may lead to a more consistent and effective control of bacterial leaf blight in the future. New sources of resistance genes should be sought, and the possibility of using induced mutations should be explored more thoroughly in developing new resistant breeding lines of rice, as suggested by Nakai and Goto (1975). It is important also to seek types of resistance other than those which involve hypersensitivity controlled by major genes. Such non-hypersensitive types of resistance may, perhaps in combination with major-gene resistance, provide the durable resistance that is so urgently needed.

Tobacco

Tobacco is attacked by several species of bacteria, but only three bacterial diseases are of great economic importance. The first of these is bacterial (or Granville) wilt, caused by *Pseudomonas solanacearum*. The second and third diseases, wildfire (caused by *Pseudomonas tabaci*) and angular leaf spot (caused by *Pseudomonas angulata*), will be considered together because the pathogens are very closely related and often occur together in the same plant. Resistant varieties play an important part in the control of all three diseases.

BACTERIAL (GRANVILLE) WILT

Bacterial wilt is widespread in parts of the USA and in Asia but it also occurs locally in Africa; the disease is restricted mainly to areas with high temperatures and very high humidity. The causal bacterium, *P. solanacearum*, has an extremely wide host range and exists in a large number of strains. The roots of susceptible tobacco plants are invaded by the pathogen, which attacks the conducting

tissues in particular. The main external symptoms are wilting and yellowing of leaves, especially the lower leaves, blackening and decay of the roots, and stunting. The main control measures are the use of resistant varieties and appropriate crop rotation.

Resistance to *P. solanacearum* was first discovered in 1934 in a line designated 'Tobacco Introduction No. 448A' (commonly referred to as TI 448A), which formed part of a large collection of South and Central American tobaccos screened for resistance in the USA by Clayton (1947). TI 448A was subsequently used very widely in breeding programmes as a source of bacterial wilt resistance, and in 1945 the first wilt-resistant flue-cured variety, Oxford 26, was released. Other varieties, such as Dixie Bright 101 and 102, and Coker 139, later combined resistance to bacterial wilt with resistance to black shank, a serious fungal disease; many bacterial wilt-resistant varieties were also resistant to *Fusarium* wilt. These early resistant varieties were usually of lower quality than older, more susceptible varieties but they were soon superseded by resistant varieties with higher quality, for example NC 73, NC 75 and NC 95 in North Carolina (Moore *et al.*, 1962).

Breeding programmes were started in other parts of the world following these successful pioneering investigations in the USA. For example, Schweppenhauser (1966) reported that TI 448A, Oxford 26, and Dixie Bright 101 and 102 show good resistance to *P. solanacearum* in Rhodesia also, and Coker 139 has been used as a source of resistance in Japan (Nakamura, 1967) and Taiwan (Chang and Lai, 1968). In the Philippines, crosses have been made between introduced resistant tobaccos, such as TI 448A and Oxford 26, and susceptible native varieties (Gutierrez and Figueroa, 1967). Progenies of crosses between Oxford 26 and Vizcaya, and between TI 448A and Vizcaya were almost as resistant as the more resistant parent, suggesting that resistance is a partially dominant character. This supports the results of Schweppenhauser (1966) who also concluded from hybridization experiments that resistance to *P. solanacearum* is dominant but not simply inherited. Conversely, Smith and Clayton (1948) in the USA and Nakamura (1967) in Japan have reported that resistance is recessive and polygenically controlled. The reasons for these conflicting reports are uncertain, but the situation has a parallel in bacterial blight of cotton where Arnold (1963) suggested that confusion regarding the inheritance of resistance to *Xanthomonas malvacearum* might be explained by a very strong genotype—environment interaction (*see* page 181). Resistance to *P. solanacearum* in tobacco may be influenced by environment to a similar extent, giving rise to different conclusions regarding the inheritance of resistance, in different countries.

There are several races of *P. solanacearum*, each distinguished by their ability to infect certain plant species. For example, isolates of race 1 are pathogenic to tobacco; those of race 2 are avirulent on tobacco but pathogenic to triploid bananas and those of race 3 are pathogenic to potato but not to tobacco (Kelman and Sequeira, 1972). There is considerable variation between isolates of the same race, but the importance of this variation in breeding for resistance to bacterial wilt in tobacco is uncertain. There does not appear to be any published record of races that are able to overcome the resistance of such varieties as TI 448A, Oxford 26 or NC 95. Apparent breakdowns of resistance in the field have been attributed to the presence of unusually high concentrations of inoculum, and to exceptionally favourable conditions for spread of the disease, rather than to resistance-breaking strains of *P. solanacearum* (e.g. Granada and Sequeira, 1975).

The nature of resistance to bacterial wilt is not clearly understood although resistance seems often to be expressed as a hypersensitive response. Resistant varieties often show stunting or dwarfing but usually recover from wilt (Akehurst, 1968) which implies, either that the roots of resistant plants are less affected by the disease, or that the translocation system continues to operate effectively in spite of infection. Because plants that are resistant to bacterial wilt are frequently resistant also to *Fusarium* wilt, there may be a common resistance mechanism which is effective against both diseases. If this is so, such a mechanism is probably non-specific in its action; resistance-breaking races of *P. solanacearum* should not, therefore, be a serious problem. Certainly, many resistant varieties have given a good control of bacterial wilt over many years in several different tobacco-growing areas of the world.

WILDFIRE AND ANGULAR LEAF SPOT

Wildfire, and angular leaf spot, which are caused by two very closely related species of *Pseudomonas*, are found in all the major tobacco-producing areas of the world. The disease caused by *P. tabaci* can spread very rapidly (i.e. 'like wildfire') in susceptible crops under conditions of high humidity. Characteristic symptoms of wildfire are necrotic spots, each surrounded by a yellow halo, on the leaves; under suitable conditions the lesions enlarge rapidly and coalesce, making the leaves die prematurely. Symptoms of angular leaf spot are similar to those of wildfire, except that the necrotic spots have an angular appearance and do not have halos.

These diseases can be partly controlled by appropriate seed dressings, certain cultural practices and sprays of copper or streptomycin. Several resistant varieties, mainly Burley types, have been released in the USA and have contributed to the control of wildfire and angular leaf spot (Akehurst, 1968). Although breeding work has been concentrated on resistance to *P. tabaci*, resistance to one pathogen is usually closely associated with resistance to the other.

Good sources of resistance to *P. tabaci* and *P. angulata* have not been found in *Nicotiana tabacum*, but many other species of *Nicotiana* are resistant, and interspecific breeding programmes have been carried out to transfer this resistance to cultivated types. For example, a cross between tetraploid *N. tabacum* and tetraploid *N. longiflora* gave rise to a resistant breeding line, TI 106, which was then backcrossed to various tobacco varieties (Clayton, 1958); highly resistant lines with acceptable levels of quality and yield were obtained when White Burley was used as the recurrent parent. Heggstad *et al.*, (1960) developed the first wildfire-resistant variety, Burley 21, which was released in the late 1950s for use in Tennessee. This was later replaced by other wildfire-resistant varieties, such as Burley 37 and Burley 49, which were superior to Burley 21 in several respects, including resistance to other diseases. Burley 21 has been used as a source of resistance to *P. tabaci* in Rhodesia, where it has been crossed with two tobacco varieties which have shown tolerance to wildfire, namely Virginia Gold and Meadows Giant (Raeber, 1966; Raeber and Smeeton, 1970).

Nakamura and Nakatogawa (1965) evaluated 162 *N. tabacum* varieties and 17 wild *Nicotiana* species for resistance to *P. tabaci* under Japanese conditions, and found that line TI 106 and Burley 21 were highly resistant; three Chinese varieties and one from Rhodesia were moderately resistant, and the remainder

showed varying degrees of susceptibility. Of the wild species, *N. rustica*, *N. longiflora*, *N. plumbaginifolia*, *N. repandra*, *N. alata*, *N. affinis*, *N. sanderae*, *N. forgetiana* and *N. acuminata* showed high levels of resistance. Oganesyanyan (1969), working in Armenia (USSR), where *P. tabaci* is a serious pathogen, has also reported that *N. plumbaginifolia* is a potentially important source of resistance. Resistance to *P. tabaci* has been successfully transferred to *N. tabacum* from *N. repandra* (Pittarelli and Stavely, 1975).

Valleau, Litton and Johnson (1962) found strains of *P. tabaci* and *P. angulata* to which Burley 21 and other varieties carrying the resistance factor from *N. longiflora* are susceptible. However, it is not known how widespread these resistance-breaking races are, nor what threat they constitute to the future successful control of wildfire by resistant varieties. There seem to have been no serious or widespread breakdowns of resistance in any variety by new physiologic races of either pathogen.

There is little published information concerning the inheritance or nature of resistance to wildfire or angular leaf spot. When a resistant line, TI 106, was crossed with some susceptible tobaccos, resistance did not segregate in Mendelian ratios, although such ratios were obtained in progenies of crosses with White Burley (Akehurst, 1968). The reasons for these different results are not understood, but genes in some tobacco varieties may modify the expression of resistance to *P. tabaci* and *P. angulata* derived from *N. longiflora*. It is not clear what types of resistance mechanisms are involved, but hypersensitivity seems to be implicated. Lovrekovich, Lovrekovich and Stahman (1967) found a correlation between high peroxidase activity in mature tobacco leaves and resistance to *P. tabaci*, but there is no evidence of a direct causal relationship between these two factors.

Resistant tobacco varieties play an important part in the control of bacterial wilt, wildfire and angular leaf spot in many parts of the world. Wilt-resistant varieties have been grown successfully for many years, and resistance-breaking races of *P. solanacearum* have not been an important problem. Although races of *P. tabaci* and *P. angulata* that can overcome resistance to *N. longiflora* have been known for many years, these have not severely attacked resistant varieties. Resistance to all three diseases is controlled by a small number of genes and can therefore easily be managed in breeding programmes. This has enabled plant breeders to produce varieties that are resistant to several of the most important pests and diseases of tobacco, including bacterial wilt, downy mildew, *Fusarium* wilt and nematodes (Rogers, 1975).

Tomato

Attempts have been made to breed for resistance to two serious bacterial diseases of tomato: wilt and canker.

BACTERIAL WILT

Bacterial wilt, caused by *Pseudomonas solanacearum*, is a very serious disease of tomato crops in many tropical and subtropical regions, where it is often one of the most important yield-limiting factors. It is a very difficult disease to control by chemical or cultural methods and, accordingly, there have been many programmes of breeding for resistance.

Much of the early breeding work was carried out in North Carolina (Schaub and Bayer, 1944; Weaver, 1944). In field tests, Louisiana Pink and a

Lycopersicon esculentum line, T414 (accession no. PI 3814) from Puerto Rico showed good resistance; crosses between these two tomatoes were considered to be particularly promising sources of resistance to bacterial wilt. Aberdeen (1946) tested a number of tomato varieties for resistance to bacterial wilt in Australia, and found that strains derived from Louisiana Pink and T414 were resistant also in Queensland; furthermore, he found that Sensation and Marvel showed good resistance to *P. solanacearum*, but the fruit of these resistant varieties was of poor quality. Resistant lines from North Carolina were found to be resistant in Hawaii also (Anonymous, 1951) but their fruits were considered to be too small to be of much commercial value.

Abeygunawardena and Siriwardena (1963) tested 49 tomato varieties and hybrids for resistance to bacterial wilt in Sri Lanka. Several improved lines from North Carolina were included in these tests and four of them, together with the varieties Masterglobe and Rahangala, were the most resistant to bacterial wilt. When infection was present, resistant lines yielded much more fruit than local, susceptible varieties. However, one line, which had shown resistance to *P. solanacearum* in the Philippines, was susceptible to this pathogen in Sri Lanka; this suggested that different physiologic races of *P. solanacearum* are present in these countries. A further source of resistance, *L. pimpinellifolium* (PI 127805A), was identified in Hawaii in 1953, and attempts have been made to incorporate this resistance, which appears to be quite distinct from that previously used in North Carolina, into commercially acceptable varieties.

Many new tomato varieties with good resistance to bacterial wilt have recently been developed. For example, Venus and Saturn have been derived from crosses between Louisiana Pink and a tomato line, Beltsville 3814, in the USA (Henderson and Jenkins, 1971). These varieties, which are also resistant to race 1 of *Fusarium oxysporum*, have expressed a high level of resistance to bacterial wilt in many parts of the world including the USA (Henderson and Jenkins, 1971), the Antilles (Daly, 1973) and Taiwan (Lin, Hsu and Ho, 1974). However, Krausz and Thurston (1975) reported that Venus showed no resistance to isolate LB6 of *P. solanacearum* under conditions where this variety expressed resistance to another isolate, K60. Many tomato varieties and lines that were previously considered to be resistant in the USA and the Philippines are susceptible to isolates of *P. solanacearum* in India (Rao, Sohi and Tickoo, 1975), suggesting that resistance to bacterial wilt derived from Louisiana Pink is race-specific. However, Mew and Ho (1976) found that certain resistant tomatoes show an unexpected degree of susceptibility to bacterial wilt when they are exposed to high inoculum densities; differences in inoculum density may, therefore, explain differences in the expression of resistance by the same varieties in different areas and in different years.

The 'North Carolina' type of resistance, expressed by derivatives of Louisiana Pink, is inherited as a recessive character and is controlled by polygenes (Singh, 1961). Acosta, Gilbert and Quinon (1964) showed that resistance derived from *L. pimpinellifolium* is partially dominant in the seedling stage but is recessive in mature plants, and that the expression of resistance varies, not only with increasing age, but also with changes in temperature. It is clear, therefore, that the genetics of bacterial wilt resistance is complex.

Winstead and Kelman (1952) developed two techniques for inoculating plants with *P. solanacearum* in testing for resistance to bacterial wilt. In the first, the stems of test plants were punctured through a drop of bacterial suspension. In the second, a bacterial suspension was poured over the wounded



*Figure 6.2 The expression of resistance to bacterial wilt (*Pseudomonas solanacearum*) in tomatoes can be greatly affected by temperature. (a) BWR 1119 and BW 7580 are much more resistant to isolate LB-6 of *P. solanacearum* than Bonny Best at 26°C. (b) all three varieties are almost equally susceptible to this isolate at 32°C. (By courtesy of Dr J.P. Krausz, Pee Dee Experiment Station, Florence, South Carolina, USA)*

roots of test plants. Both techniques are equally effective in infecting susceptible tomato varieties, but root inoculations are generally considered to be better for differentiating between resistant and susceptible varieties. Lin *et al.*, (1974) inoculated plants by clipping off leaf tips with scissors that had been dipped in a suspension of bacterial cells, and obtained results which were comparable with

those from natural field infections. Although most workers have also found a good agreement between tests involving natural and artificial inoculation, the results of seedling tests do not always agree with those of adult-plant tests (Mew and Ho, 1976). The expression of resistance to bacterial blight can vary with the age of host plant (Winstead and Kelman, 1952) and with changes in temperature (Krausz and Thurston, 1975) (see Figure 6.2).

Very little is known about the mechanisms of resistance, although resistance to bacterial blight is associated with high levels of a steroidal glycoalkaloid (α -tomatine) in the roots, leaves and stems. Concentrations of α -tomatine are higher, before inoculation, in wilt-resistant parents and hybrids than in susceptible plants, and the concentration increases more in resistant than susceptible plants after inoculation (Mohanakumaran, Gilbert and Buddenhagen, 1969). High concentrations of α -tomatine are also associated with resistance to *Fusarium* wilt in tomatoes (Roddick, 1974).

The presence of certain plant parasitic nematode species in the soil can affect the susceptibility of tomato varieties to *P. solanacearum*. Temiz (1968) found that a tomato variety, Atkerson, is very susceptible to bacterial wilt even when growing in soil free of ectoparasitic nematodes, such as species of *Pratylenchus*, *Trichodorus* and *Meloidogyne*. However, some resistant varieties that did not become infected with *P. solanacearum* in the absence of nematodes became infected in soil that was infested with nematodes. There is similar interaction between nematodes and *Fusarium* wilt. In breeding for resistance to *Fusarium* and bacterial wilt, it is therefore desirable to try to combine resistance to the bacterial or fungal pathogens with resistance to nematodes. Resistance to *Meloidogyne* spp. has been reported from many sources and several resistant varieties are in cultivation.

Two serious drawbacks to the successful development of bacterial wilt-resistant tomato varieties have been that the expression of resistance is very labile and that most sources of resistance have poor quality fruit. For example, many of the North Carolina resistant lines, the Australian variety Sensation, and the *L. pimpinellifolium* derivatives, have yielded fruits that have not been of suitable size or quality for the American or Australian markets. However, commercially acceptable varieties with a moderate level of resistance are now being developed in many countries and it should be possible, eventually, to combine even more effective levels of resistance to bacterial wilt with high fruit quality and good yielding ability.

BACTERIAL CANKER

Bacterial canker of tomato, which was first recorded in the USA in 1914, occurs in many subtropical and temperate regions of the world, including North America, Europe, Australia, New Zealand and East Africa. The disease is caused by a Gram-positive bacterium, *Corynebacterium michiganense*, and is both seed- and soil-borne. The first symptom that is generally observed on an infected tomato plant is the sudden wilting of the upper parts of the plant, particularly when the fruit is beginning to ripen. This is followed by wilting of the lower leaves, which become necrotic, and brown stripes often appear on the stems; some of these stripes split open to reveal cavities in the underlying tissues. Fruits of infected plants usually become very discoloured and unmarketable,

and the disease can cause heavy losses of yield (Emmatty and John, 1973). Although partial control of bacterial canker can be achieved by seed treatment with a suitable bactericide and by crop rotation, attempts to breed resistant varieties have been made in several countries.

It was reported in 1944 that resistance to bacterial blight is present in uncultivated species of *Lycopersicon* from Peru (Anonymous, 1944). Later reports, for example those of Dimitrov (1966), confirmed that several wild tomatoes, including *L. peruvianum*, *L. pimpinellifolium* and *L. hirsutum*, show some resistance to *C. michiganense*.

Thyr (1968; 1969) tested several *L. esculentum* varieties, and several wild *Lycopersicon* species, for resistance to bacterial canker. Seedlings were raised in a glasshouse and, when they were three weeks old, the first leaf was excised; *C. michiganense* inoculum was applied to the wound with a scalpel and the reaction to inoculation was evaluated eight weeks later. Plants with one or more unwilted growing points were scored as resistant. A line of *L. hirsutum* (PI 251305) and a *L. pimpinellifolium* accession from Peru showed high levels of resistance, and plants of *L. glandulosum* and *L. peruvianum* were found to have moderate resistance. In other tests, Thyr recorded the proportion of surviving plants in three lines of *L. esculentum* and two of *L. pimpinellifolium* after inoculation with *C. michiganense* at the seedling stage. The two *L. pimpinellifolium* lines had 87 and 92 per cent of surviving plants, respectively, eight weeks later. Two of the tomato lines, Bulgarian 12 and Utah 659, that had shown resistance in previous tests, had 83 per cent and 52 per cent of survivors respectively, compared with only 7 per cent in the susceptible *L. esculentum* control variety (PI 283907). In the field, with natural infection, this susceptible variety was much more badly damaged by bacterial canker than were the lines Bulgaria 12 and Utah 659. The resistance of the variety Bulgaria 12 to bacterial canker has been confirmed in a field experiment in which several tomato varieties were inoculated with two isolates of *C. michiganense*; yield reductions in these varieties ranged from none in Bulgaria 12, to 39 per cent in the most susceptible variety (De Jong and Honma, 1976a).

Many different screening techniques and methods of assessing resistance to *C. michiganense* have been developed (De Jong and Honma, 1976a). For example, Hassan, Strider and Konsler (1968) sprayed the cotyledons of tomato plants with an aqueous suspension of *C. michiganense* cells, and susceptibility was assessed by comparing the numbers of bacteria-induced spots that developed on the cotyledons in different varieties. The results of these tests agreed with those of tests involving root and stem inoculation. All the varieties of *L. esculentum* and all wild *lycopersicon* species except *L. hirsutum* (PI 251305), which were tested in this way, were susceptible, but to different degrees. Many workers, including Lazăr and Bucur (1961), Dimitrov (1966) and Elenkov (1965) have reported that some tomato varieties show moderate levels of resistance to bacterial canker. Ercolani (1967) reported that Fiortenina, which was at one time widely grown in the Po Valley of Italy, is resistant to bacterial canker, another variety, Roma, is very susceptible. Of 14 tomato varieties which were commonly grown in Japan in 1968, one (Sekko) was found by Wakimoto, Uematsu and Mukoo (1968) to be resistant to *C. michiganense*.

There is little published information about the genetics of resistance to bacterial canker. Kuriyama and Kuniyasu (1974) reported that resistance derived from *L. hirsutum* var. *glabratum* is controlled by multiple genes and is expressed

as an incompletely dominant character. The resistance of *L. pimpinellifolium* to a 'mild strain' of *C. michiganense* is also controlled by several genes, including a recessive gene (*a*) and three dominant genes (*B*, *C* and *D*); three alleles of *C* and *D* genes have been identified (De Jong and Honma, 1976b). The resistance of *L. hirsutum* apparently involves an additional recessive gene *x*, homozygous plants (*xx*) being resistant to bacterial canker only where gene *C* and a modifying gene *F* are present.

Seed transmission of *C. michiganense* is more common in some tomato varieties than others. For example, seed transmission frequencies in Highlander, Heinz 1350 and Campbell 28 were 49, 3 and 0 per cent respectively (Thyr *et al.*, 1973). This raises the possibility of breeding varieties with a low transmission frequency of the pathogen through the seed. This resistance would reduce the need for chemical seed dressings and would enhance the effectiveness of other types of resistance.

No resistance-breaking races of *C. michiganense* have been reported; resistance to bacterial canker would, therefore, seem to be durable. Thyr (1972) tested a number of tomato varieties and accessions of *L. pimpinellifolium* and *L. hirsutum* against a range of isolates collected from different parts of the USA, and found differences in aggressiveness between *C. michiganense* isolates, but no evidence of specific interactions between isolates and individual host genotypes.

It can be concluded that useful levels of resistance to bacterial wilt and bacterial canker exist in certain tomato varieties and in several wild species of *Lycopersicon*, and that these have been used to produce many resistant varieties. Some of these varieties have been grown extensively and have helped to reduce losses from these diseases in several of the main tomato-growing areas of the world. Unfortunately, resistance to bacterial wilt is often associated with low yields of poor quality fruit, and seems to be race-specific; there is, therefore, a danger that resistance-breaking races of *P. solanacearum* may become widespread. It should be possible to develop high-yielding, high-quality varieties which express greater resistance to both diseases than present resistant varieties. It is unlikely, however, that resistant varieties by themselves will give an adequate, long-term control of either bacterial wilt or bacterial canker.

Pears and Apples

FIREBLIGHT

Fireblight of pears was the first plant disease shown to be caused by a bacterium (*Erwinia amylovora*). This disease causes severe losses in pear crops in many parts of the world; although *E. amylovora* can infect apples and plums, losses in these crops are usually less serious than in pears. Because apples are generally more resistant to fireblight than are pears, they can be grown in many areas where pears cannot (Eden-Green and Billing, 1974). Pollinating insects convey the bacteria from flowers of infected trees to those of healthy trees, and other insects may spread the pathogen to shoots and leaves. Infected flowers become water-soaked and necrotic, and the young shoots may be killed; in severe infections, large branches and even whole trees may be killed. The most widespread method for control of fireblight in pears and apples is to cut out infected parts of trees, or to destroy whole infected trees by burning. Chemical control

measures, including applications of antibiotics such as streptomycin, have also been attempted (Lelliott, 1967; Van Der Zwet, 1970), but streptomycin-resistant strains of *E. amylovora* have been identified (Moller *et al.* 1973). However, none of these measures has very effectively controlled fireblight in orchards, and attempts have been made to find resistant varieties of both pears and apples. It has been realized for many years that pear and apple varieties differ greatly in their susceptibility to fireblight, although no commercial variety is resistant under all conditions (Eden-Green and Billing, 1974).

Pears

Many collections of pear (*Pyrus*) varieties have been screened for resistance to fireblight, particularly in the USA (e.g. Brooks and Olmo, 1958; Cameron, Westwood and Lombard, 1969; Van Der Zwet, Oitto and Blake, 1974) and in Canada (e.g. Lockhart and Gourley, 1966; Layne, Bailey and Hough, 1968). Oitto, Van Der Zwet and Brooks (1970) tested more than 500 pear varieties from various countries and found that most were very susceptible to *E. amylovora*. In these tests, only nine varieties showed no fireblight symptoms when exposed to infection although a further 45 varieties were classified as highly or moderately resistant. Van Der Zwet *et al.*, (1974) tested 20 pear varieties for fireblight resistance in six successive years in natural disease epidemics and nine of these varieties, including Magness and Maxine, were rated as resistant. Layne *et al.*, (1968) had previously reported that Magness and Maxine were among the more resistant pear varieties that they had tested in Canada.

Several uncultivated species of *Pyrus* show much higher levels of resistance to fireblight than varieties of *P. communis*, and these have been used as sources of resistance. The best of these sources are certain clones of *P. ussuriensis*, *P. serotina*, *P. calleryana* and *P. pyrifolia* (Oitto, 1967; Layne *et al.*, 1968; Van Der Zwet, Oitto and Westwood, 1974). Crosses have been made between high-quality susceptible pear varieties and resistant *Pyrus* species, and some selections from them yield high quality fruit and express good resistance to *E. amylovora*; these are being used in breeding programmes. Although Bell *et al.* (1976) found a negative correlation between resistance to fireblight and high quality fruit in the progenies of some crosses, it has not generally been difficult to select both for resistance and high quality.

Techniques of evaluating resistance to fireblight in pears have been compared by Thompson, Janick and Williams (1962). The most widely used method is to encourage field epidemics of fireblight by interplanting susceptible pear trees among those to be tested. These susceptible trees can be inoculated, if necessary, to start an epidemic. The severity of disease symptoms on individual trees can then be assessed by eye, using a rating system such as that used by Thompson, Zimmerman and Van Der Zwet (1975). Seedling tests have also been devised; Van Der Zwet and Zook (1976) inoculated 100-day-old seedlings and assessed the severity of fireblight symptoms on them about four weeks later.

Greenhouse tests have also been used to screen pears for resistance to fireblight (Quamme, Van Der Zwet and Dirks, 1976). Such tests distinguished between very resistant and very susceptible varieties, but varieties were frequently ranked for susceptibility in a different order in greenhouse and field tests. Greenhouse tests with seedlings are therefore unlikely to supplant field tests completely, but will enable very large numbers of plants to be tested quickly and easily.

Layne *et al.* (1968) could draw no definite conclusions about the inheritance of resistance to fireblight in pears, from progeny tests of either interspecific or intraspecific crosses. In some crosses resistance was recessive and appeared to be controlled polygenically while, in others, resistance was a partially dominant character. Later work suggested that two dominant genes, *Ew*₁ and *Ew*₂, condition resistance to fireblight in *P. pyrifolia* and *P. ussuriensis* respectively. Thompson *et al.* (1975) have identified a single dominant gene in pears, *Se*, the presence of which conditions sensitivity to fireblight that is so severe that infected trees are killed. Of 24 pear varieties and selections tested, 17 were heterozygous for the *Se* gene (i.e. *Se se*) and 7 were homozygous (*se se*) trees; this latter group showed a high level of resistance to *E. amylovora*. Some trees that were heterozygous at the *Se* locus transmitted more resistance to their progeny than others (Thompson, 1976); this suggests that other, modifying genes are also involved. Heritability estimates from 256 crosses indicated that about one-half of the variability is additive (Bell *et al.*, 1977), which in turn suggests that several resistance genes are involved. Evidence for non-additive variance was compatible with the assumption that a gene for sensitivity (*Se*) is widespread in pears. None of the cultivars tested by Thompson (1976) were homozygous *Se Se* trees but certain other varieties, including Aurora, Forelle and De Voe, probably are homozygous. The elimination of the dominant gene *Se* from breeding material should enable plant breeders to avoid the development of new, very susceptible pear varieties in future (Thompson *et al.*, 1975).

Little has been written about the nature of resistance to fireblight in pears, but resistance to infection seems mainly to be implicated. For example, Van Der Zwet, Keil and Smale (1969) recorded large varietal differences in the number of trees which became infected under natural conditions in Arkansas in 1967. Bartlett, which is widely recognized as being very susceptible to fireblight, had the highest percentage (95 per cent) of infected trees, whereas no trees of a resistant variety, Maxine, showed signs of infection. Over 80 per cent of trees of Magness, which had previously been considered to be a resistant variety in other parts of the USA, were infected; this was the first record of severe fireblight in this variety. This apparent susceptibility could have been caused by unusual environmental conditions, or by the presence of a resistance-breaking race of *E. amylovora*. However, older wood of Magness is now known to be very susceptible to fireblight, although young wood is resistant (Eden-Green and Billing, 1974); this differential effect of age on the expression of resistance probably accounts for some of the conflicting reports about the level of fireblight resistance expressed by this and other varieties. There is no convincing direct evidence about the existence of resistance-breaking races of *E. amylovora*.

Hildebrand, Powell and Schroth (1969) found that fireblight-resistant pear varieties and *Pyrus* species often contain higher concentrations of an antibiotic substance, arbutin, than susceptible types. However, no causal relationship between arbutin, β -glucosidase or phenolic compounds and resistance has been established (Hildebrand *et al.*, 1969; Challice and Westwood, 1972).

Apples

Apple varieties differ greatly in their susceptibility to fireblight. For example, Boyce (1970) found that more trees became infected with *E. amylovora* in Mutsu (= Crispin) than in McIntosh or Delicious in natural-infection field trials

in the USA. The resistance of these two latter varieties and Golden Delicious has recently been confirmed in field trials in three climatically distinct areas of the USA and Canada (Aldwinckle, 1974). More than 1000 apple varieties and selections of *Malus* spp. have recently been screened for resistance to fireblight in the USA and many potentially useful sources have been identified (Aldwinckle *et al.*, 1976). In addition, several dwarfing rootstocks, including the Malling rootstocks, M2, M7 and M15, and the Malling-Merton rootstocks, M104, M105, M110 and M111, have shown good resistance to *E. amylovora* at two sites in the USA (Keil and Van Der Zwet, 1975). Tolerance has been identified in the variety Haralson (Lake, Stushnoff and Kennedy, 1975).

Although testing for fireblight in apples has usually involved field observations in natural disease epidemics, the results of glasshouse tests with young trees usually agree with those of field experiments (Aldwinckle and Cummins, 1974). There is very little published information on the genetics of fireblight resistance in apples. Most progenies of crosses between susceptible and resistant commercial rootstocks, varieties, selections and *Malus* spp. have shown intermediate levels of resistance (Aldwinckle and Cummins, 1974). However, resistance was mainly dominant in progenies of crosses between *M. prunifolia* × *M. sieboldii* hybrids and apple varieties. It seems, therefore, that there are at least two different types of resistance. The flowers of resistant varieties contain higher concentrations of catecholases, cresolases and phenolic compounds than do those of susceptible varieties (Ahn and Stushnoff, 1973). However, evidence for a causal relationship between these compounds and resistance to fireblight is circumstantial.

Some existing pear and apple varieties show significant levels of resistance to fireblight, and these varieties can be used in areas where epidemics of the disease can be expected. Several programmes of breeding for resistance in both pears and apples, using cultivated varieties and wild *Pyrus* and *Malus* species as sources of resistance, are in progress, particularly in the USA and Canada. There does not seem to be much difficulty in developing resistant varieties with high yields and high quality fruit, and such varieties should eventually play a very important part in reducing fireblight damage in apples and pears. In the short term, however, eradication of diseased trees, supplemented by chemical control measures where appropriate, will continue to be the most important method of controlling fireblight.

References

The references cited in this chapter, together with those for Chapters 5, 7, 8 and 9, are listed in References – Part III, pages 267–290.

RESISTANCE TO DISEASES ASSOCIATED WITH MYCOPLASMA-LIKE ORGANISMS (MLO) AND RICKETTSIA-LIKE ORGANISMS (RLO)

The Importance of Plant Diseases Associated with MLO and RLO

It now appears that many yellows-type diseases of crop plants are not caused by viruses, as was thought until recently, but by micro-organisms, particularly mycoplasma-like organisms (MLO) and rickettsia-like organisms (RLO). Doi *et al.* (1967), and Ishiie *et al.* (1967) found micro-organisms, which could be controlled by tetracycline antibiotics, in the phloem tissues of plants showing symptoms of mulberry dwarf, potato witches' broom and aster yellows. Their discoveries stimulated much research into the identity and properties of these micro-organisms.

Table 7.1 SOME IMPORTANT PLANT DISEASES THAT ARE ASSOCIATED WITH MYCOPLASMA-LIKE AND RICKETTSIA-LIKE ORGANISMS (AFTER MARKHAM, 1978)

<i>Type of Pathogen and Disease</i>	<i>Plant hosts</i>	<i>Distribution</i>	<i>Vector</i>
Mycoplasma-like organisms			
Rubbery wood	Apple	Europe	?
Aster yellows	Many crop plants	America, Europe, Asia	Leafhoppers
Citrus stubborn	Citrus crops	America, Africa	Leafhoppers
Clover phyllody	Clover, strawberry	N. America, Europe	Leafhoppers
Lethal yellowing	Coconut	N. America, West Indies	?
Corn stunt	Maize	N. America	Leafhoppers
Pear decline	Pear	N. America	Psyllids
Grassy stunt	Rice	Asia	Leafhoppers
Yellow dwarf	Rice	Asia	Leafhoppers
Yellow wilt	Sugar beet	S. America	Leafhoppers
Yellow stunt	Sorghum	N. America	?
Rickettsia-like organisms			
Pierce's disease (alfalfa dwarf)	Many crops including grape	N. America	Leafhoppers and froghoppers
Clover club leaf	White clover	Europe	?
Phony disease	Peach	N. America	Leafhoppers
Ratoon stunt	Sugar cane	S. America, Australia	?
Apple proliferation	Apple	Europe	?

About 60 plant diseases comprise the group known as yellows-type diseases, some of which are listed in *Table 7.1*. Infected plants are stunted and often deformed, have a characteristic upright growth habit and commonly show chlorosis and vein-clearing symptoms on the leaves (Maramorosch, Shikata and Granados, 1968). Most of the known MLO that are associated with yellows-type diseases in plants are very similar in morphology and structure to those MLO that cause diseases in higher animals (*see Figure 7.1*). At least two other diseases

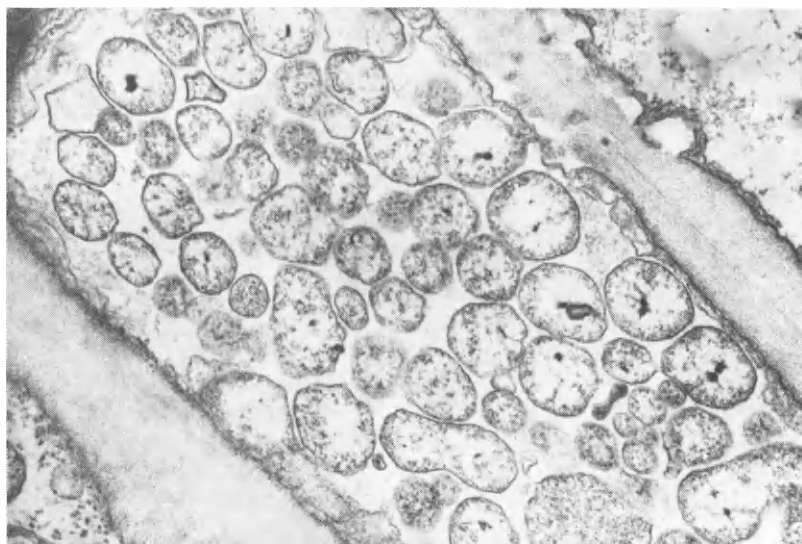


Figure 7.1 Cells of the clover phyllody MLO in the phloem of infected white clover are pleiomorphic and vary in diameter from 60–650 nm. (By courtesy of Dr P.R. Markham, John Innes Institute, Norwich)

are associated with spiroplasmas, which are helical, motile MLO (*see Figures 7.2 and 7.3*) and more than 17 plant diseases are associated with RLO (Hopkins, 1977; Markham, 1978). Many of these diseases can cause serious losses of yield in crop plants. Most are transmitted by leafhoppers (Homoptera, Cicadellidae) or froghoppers (Homoptera, Cercopidae). Pear decline, which is associated with an RLO, is transmitted from tree to tree by species of Psyllids (Homoptera, Psyllidae). Aster yellows, which can attack a wide range of different crop plants, is one of the most destructive plant diseases (Chapman, 1974). In the USA, for example, total crop losses due to aster yellows have been reported in carrots, potatoes and celery, and lettuce and spinach crops have also been extensively damaged. The disease has also caused serious losses in Canada where, in addition to these crops, flax, rapeseed, sunflower and buckwheat have been severely damaged.

Most yellows-type diseases, however, have a restricted host range and their damage is usually confined to one, or a few, crop plant species. They can, nevertheless, be of considerable economic importance. For example, yellow wilt has devastated sugar beet crops in Argentina (Bennett and Munck, 1946) and in Chile (Bennett *et al.*, 1967) for many years and is undoubtedly one of

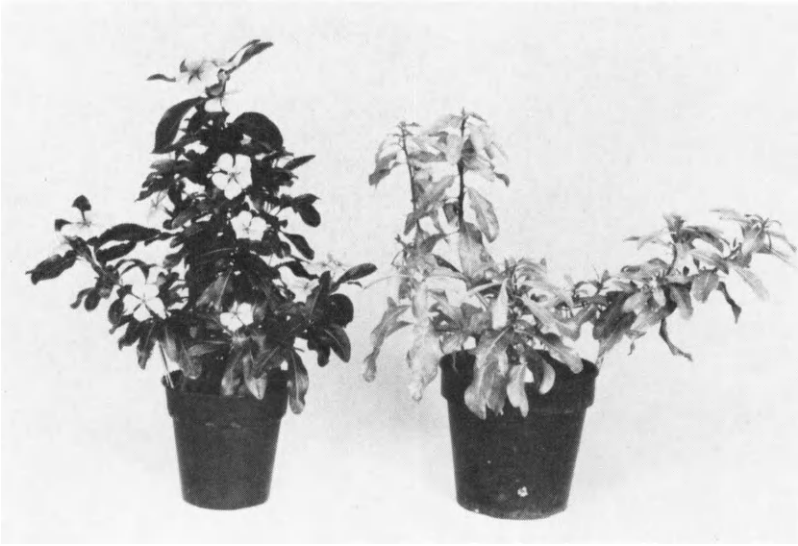


Figure 7.2 Citrus stubborn is a severe yellows disease affecting citrus crops and Vinca rosea (periwinkle). It is associated with Spiroplasma citri, a MLO with a characteristic spiral shape. (By courtesy of Dr P.R. Markham, John Innes Institute, Norwich)

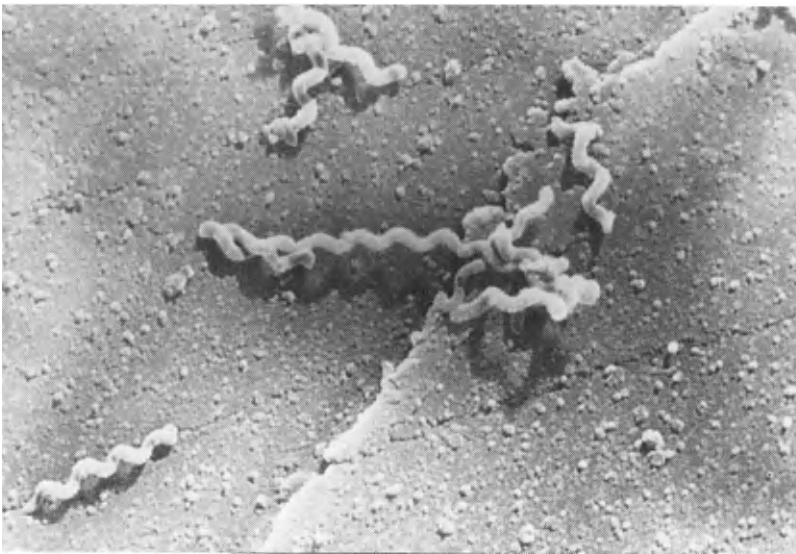


Figure 7.3 Scanning micrograph of Spiroplasma citri cells which vary in size from 1–15 μm in length and are about 0.2 μm in diameter. (By courtesy of Dr P.R. Markham, John Innes Institute, Norwich)

the most destructive diseases of sugar beet. Corn stunt, which is known to be caused by a spiroplasma (*Figure 7.4*), is one of the most serious diseases of maize in the south-eastern parts of the USA and can decrease yields of grain by more



Figure 7.4 Corn stunt spiroplasma in the phloem of infected maize. (By courtesy of Dr P.R. Markham, John Innes Institute, Norwich)

than 20 per cent. Coconut palms that become infected with lethal yellowing usually die within six months after inoculation and this disease has caused considerable losses, particularly in the West Indies. Pierce's disease, also known as alfalfa dwarf, which is associated with an RLO, can attack many crop plants and has caused significant yield reductions in grapes and alfalfa in parts of the USA. Sugar cane and apple are damaged by ratoon stunt and apple proliferation respectively, which are also RLO-associated diseases.

Some Characteristics of MLO and RLO

For a detailed account of MLO and RLO as plant pathogens, the reader is referred to one of several excellent reviews, for example that of Maramorosch (1974). A brief account only will be given here of some of the more important features of these micro-organisms and of the diseases that they cause.

Mycoplasmas are highly pleiomorphic micro-organisms which are surrounded by a characteristic triple-layered membrane, although they lack a rigid cell wall. They are the smallest known organisms capable of an independent existence. Like bacteria, but unlike viruses, they can be cultured on cell-free media but only a few, for example corn stunt MLO (Williamson and Whitcomb, 1975), the citrus little-leaf spiroplasma (Markham *et al.*, 1974) and the citrus stubborn MLO (Fudl-Allah, Calavan and Igwegbe, 1971) when cultured in this way have been transmitted to host plants. Koch's postulates to prove pathogenicity have not yet been satisfied for most of the other potential MLO and RLO pathogens. MLO, unlike most bacteria, are unaffected by penicillin but they are very sensitive to tetracycline antibiotics, and tetracyclines have been used to control MLO-associated diseases in the field. Conversely, RLO are very sensitive to penicillin but are quite insensitive to tetracyclines.

All MLO that are associated with yellows-type diseases have many characteristics in common. For example, most are transmitted by leafhoppers in which they have long incubation periods, after which they persist for long periods in the vector, often for life. They are not, however, passed through the egg to the progeny of infected leafhopper vectors. When mycoplasmas are introduced into a host plant they have a long incubation period and are confined to phloem



Figure 7.5 The flowers of white clover plants infected with clover club leaf are very stunted (left) compared with those of healthy plants (right). (By courtesy of Dr P.R. Markham, John Innes Institute, Norwich)



Figure 7.6 The phloem of clover infected with club leaf contains Rickettsia-like organisms (RLO) with cells approximately 1–2 μ m long and 0.2 μ m in diameter. (By courtesy of Dr P.R. Markham, John Innes Institute, Norwich)

cells. They have been transmitted from plant to plant by grafting and by species of dodder, but not by mechanical inoculation.

Two yellows-type diseases, corn stunt and citrus stubborn, are caused by helical, prokaryotic wall-less micro-organisms for which the term 'spiroplasma' has been adopted (Saglio *et al.*, 1973). Although spiroplasmas are morphologically distinguishable from other mycoplasmas they are very similar in most respects.

Plant pathogenic RLO differ from MLO in several important characteristics. Rickettsiae are small, coccoid, rod-shaped or pleiomorphic, Gram-negative, intracellular parasites which are usually much larger than mycoplasmas (see *Figures 7.5 and 7.6*). In contrast to mycoplasmas, RLO are usually bounded by a cytoplasmic membrane and a well-defined cell wall (Davis and Whitcomb, 1971). Although many RLO are pathogens of insects and other arthropods, only 12 are known to be plant pathogens. No vector has been identified with several of these pathogens but the remainder are transmitted either by leafhoppers, which feed on the phloem of host plants, or by froghoppers which are mainly xylem feeders. Some RLO are confined to the phloem of the host plant and others to the xylem, depending on the feeding behaviour of the specific vectors concerned. Unlike mycoplasmas, many RLO are passed on from one generation of the vector to the next through the eggs.

The Epidemiology of Diseases Associated with MLO and RLO

The natural vectors of many MLO or RLO are not known and little information is available concerning the epidemiology of the diseases with which they are associated: it is probable, however, that most of these pathogens will eventually be shown to be transmitted by specific insect vectors. Many MLO and RLO in vegetatively propagated crops are spread by means of diseased propagating material because they can readily be transmitted by grafting and in cuttings. In such diseases vector transmission may be of secondary importance only.

Until 1968, it was thought that there were two main types of leafhopper-transmitted plant viruses: first, those which multiplied in the vector, in which they were often pathogenic; second, those which did not multiply in the vector. It is now generally accepted that pathogens of the first group of so-called 'viruses' are MLO or RLO rather than true viruses. However, because the transmission characteristics of MLO and RLO are very similar to those of persistent, insect-transmitted viruses, such as curly top virus, the epidemiology of the diseases that they cause is also similar. Much of what has been written about the epidemiology of persistent insect-transmitted viruses (see page 215) is applicable also to insect-transmitted MLO and RLO.

Methods of Disease Control

Methods which have been used to control diseases caused by, or associated with, MLO and RLO are very similar to those used against insect-transmitted virus diseases. These methods include several cultural practices, such as manipulation of the time of planting or spacing of the plants within a crop, and the removal of potential sources of infection. The removal of infected weed hosts or volunteer plants can greatly reduce the disease potential. In vegetatively propagated crops

it is obviously important to propagate only from disease-free material. With high-value crops, diseased propagating material can be treated with appropriate antibiotics or heat to rid it of pathogenic MLO and RLO (Markham, 1978). Antibiotics have also been used commercially in the therapy of pear decline disease: two or three annual treatments with oxytetracycline hydrochloride restored diseased trees to near-normal condition (Nyland and Moller, 1973).

The most widely practised control method has been the treatment with insecticides of crops threatened with insect-transmitted MLO and RLO diseases; this reduces the numbers of vectors that are present to spread disease. Vectors have also been sprayed with insecticides on their breeding grounds on natural vegetation to reduce the threat to nearby crops in the subsequent growing season. Under suitable conditions, several systemic insecticides, applied either to the foliage or to the soil, have given a satisfactory control of vectors and consequently of the spread of insect-transmitted MLO or RLO pathogens. However, frequent applications of insecticides are often necessary to achieve a good control and this can be laborious and expensive. The development of integrated control programmes is therefore a matter of urgency, and varieties that are resistant either to MLO or RLO pathogens or to their insect vectors are important components of such programmes.

Control by Resistant Varieties

Heritable resistance to MLO and RLO has been demonstrated in several crops, and progress in breeding for resistance to some important diseases will be reviewed briefly in this section.

Some lettuce and aster varieties become infected with aster yellows much less readily than others (Chapman, 1974). For example, in a field experiment, 30 per cent of plants of one lettuce variety became infected with aster yellows compared with over 60 per cent of another. Some carrot varieties also have shown good resistance to aster yellows, based partly on resistance to the pathogen itself and partly on resistance to the leafhopper vector. There are excellent prospects for developing varieties that are very resistant to aster yellows in most of the crops at risk.

Corn stunt *spiroplasma* can be transmitted by several species of leafhoppers. Although heritable resistance to this disease was first identified in Texas in 1945, very few high-yielding resistant hybrids have yet been marketed. Considerable progress has been made recently, however, with the incorporation of corn-stunt resistance into high-yielding breeding material. Selection of resistant parents on the basis of data from replicated tests greatly improved the level of resistance in just one generation. For example, after one cycle of selection for resistance, only 21 per cent of progenies from selected plants became infected with corn stunt under conditions in which 52 per cent of the original population became diseased (Scott and Rosenkranz, 1974a). Several promising sources of resistance, including the maize population designated Kenya Composite, have been found in the USA (Scott and Rosenkranz, 1975).

Nelson and Scott (1973) studied the inheritance of resistance to corn stunt and showed that general combining ability is more important than specific combining ability, additive gene action being much more important than the dominance of gene action. In a diallel analysis experiment the level of resistance

expressed by hybrids between resistant and susceptible parents was intermediate between that shown by progenies of resistant or susceptible parents. Later work by Scott and Rosenkranz (1974b) led to the conclusion that many genes are involved in the control of resistance, on the basis of data obtained from F_2 progenies of crosses between resistant and susceptible plants. Scott and Rosenkranz also showed that resistance to corn stunt and maize dwarf mosaic virus disease are independently inherited, although previous work had suggested that these resistances were genetically linked.

Varietal differences in resistance to the rubbery wood disease have been reported in some apple and pear varieties. For example, the apple varieties Lord Lambourne and Victory were more sensitive to rubbery wood than were 20 other varieties tested (Dhingra and Ahlawat, 1973). In inoculation tests involving the grafting of healthy pear scions on to diseased stocks, no symptoms of rubbery wood were observed on some varieties, for example Dekanka na Komisiyata whereas some other varieties became severely diseased (Topchińska and Topchiński, 1976). Varietal differences in the resistance of pears to pear decline have been reported by Rallo (1973), Williams and Red Bartlett being badly damaged by the disease and Mantecosa Precoz Morettini being less affected by disease than any of the varieties tested.

Coconut palms differ in susceptibility to lethal yellowing, a serious disease in the West Indies and the USA; one variety of palm, Malayan Dwarf, has been particularly resistant to the disease both in Jamaica and Florida (Midcap and McCoy, 1975). Tolerance to lethal yellowing may also be present in some varieties (Harries, 1974). Under conditions of severe natural epidemics of the disease, some infected palms in variety trials did not die within the usual period of three to six months and a few were alive after five years: laboratory tests showed that these palms were infected with the MLO that is associated with lethal yellowing. These results show that there are good prospects of breeding for resistance to lethal yellowing of coconuts.

Work in India (Narayanasamy and Jaganathan, 1973) and China (Lin, Chu and Chien, 1975) has shown that some varieties of rice are more resistant to yellow dwarf than others. Muniyappa and Ramakrishnan (1976) inoculated seedlings of 34 rice varieties with the yellow dwarf pathogen and found that the percentage of plants that became infected ranged from 4 per cent in the most resistant variety (TKM6) to 88 per cent in the most susceptible. They also found large varietal differences in resistance to yellow dwarf in the field. In Taiwan, Chen and Ko (1976a, b) artificially inoculated seedlings of 2610 varieties and lines of rice and three other *Oryza* spp. with the yellow dwarf MLO in field and glasshouse screening tests, and identified several resistant varieties.

A single dominant gene (G_s), which confers resistance to grassy stunt, has been transferred to cultivated rice (*Oryza sativa*) from a related species, *Oryza nivara*. Resistance to the leafhopper vector of grassy stunt, which is controlled by a gene Bph_1 , has been combined with this resistance to grassy stunt in high-yielding breeding lines at IRRI (Khush and Ling, 1974). Resistance to grassy stunt and its leafhopper vector, *Nilaparvata lugens*, has also been found in a local Indian rice variety, Triveni (Kulshreshtha, Anjaneyulu and Padmanabhan, 1974).

The clover phyllody MLO can affect both clovers (Figure 7.7) and strawberries. Strawberry varieties differ greatly in resistance to clover phyllody (strawberry green petal) which is transmitted by a leafhopper, *Aphrodes bicincta*.

The most susceptible of nine varieties tested were Sparkle, Redcoat and Redchief and the most resistant were Kentville, Nova Scotia and Bibrant. The resistance of these varieties apparently involves both non-preference resistance to the leafhopper vector and resistance to the MLO associated with clover phyllody (Chiykowski and Craig, 1975).



Figure 7.7 Clover phyllody, associated with a MLO, causes the petals of white clover (and strawberry) to become leaflike. (By courtesy of Dr P.R. Markham, John Innes Institute, Norwich)

Some varieties of sweet sorghum, including Roma and Ramada, are much more resistant to yellow sorghum stunt than others such as Rio, Brandes and Sort (Zummo *et al.*, 1975). This suggests that it should be possible to produce new sorghum varieties with a high level of resistance to yellow stunt. Considerable progress has also been made in South America in breeding for resistance to yellow wilt of sugar beet (Gaskill and Ehrenfeld, 1976). Some introductions of wild sea beet (*Beta vulgaris* s.sp. *maritima*) have shown a particularly high expression of resistance to yellow wilt and these, together with other wild relatives of sugar beet, may be very useful sources of resistance. It should also be possible to improve the resistance of some sugar beet varieties by reselection.

The Nature and Inheritance of Resistance

The mechanisms of resistance to diseases that are associated with MLO or RLO have not been studied in detail; however, resistance both to the causal pathogen and to its specific vector seem often to be involved. For example, resistance to aster yellows in carrots and to clover phyllody in strawberries is based partly on resistance to the leafhopper vectors and partly on resistance to the MLO pathogens. On the other hand, resistance to corn stunt in maize seems to depend largely on some unidentified disease-escape mechanism. Tolerance seems to be a

major component of resistance to lethal yellowing in coconuts and to rubbery wood in apples.

Resistance to some MLO-associated diseases, such as corn stunt in maize, is controlled by many genes. However, resistance to other diseases, such as yellow dwarf in some rice varieties, is controlled by a single dominant gene (Lin *et al.*, 1975). Resistance to yellow dwarf is more complex in other varieties, for example Firooz-1 and Kabara, because it involves vector non-preference, a tendency to escape infection and tolerance (Chen and Ko, 1976b); such resistance is presumably controlled by many genes.

Conclusions

Significant progress has been made in developing varieties that are resistant to diseases caused by MLO or RLO, including some that are of considerable economic importance. Most of this progress was made before it was realised that these diseases were caused by micro-organisms rather than by viruses, and with little knowledge about the resistance mechanisms involved. This emphasizes that detailed information about a disease, or about the pathogens which cause it, is not an essential prerequisite of breeding for resistance. Although resistant varieties can be successfully developed by empirical methods, more information about plant pathogenic MLO and RLO, and about the vectors which transmit them, would undoubtedly improve the efficiency and effectiveness of selecting for resistance.

The fact that resistance to many diseases which are associated with MLO and RLO has been found and exploited in so many crops, should encourage plant breeders to seek higher levels of resistance to these pathogens and to search for sources of resistance to others. Even partial resistance to the diseases, or to the vectors of the pathogens, can significantly reduce the amount of damage that is caused by MLO and RLO. Partial resistance can also decrease the amount of insecticides that must be applied to protect crops from insect-transmitted pathogenic MLO and RLO. Resistant varieties will play an increasingly important part in the control of MLO- and RLO-associated diseases in the future.

References

The references cited in this chapter, together with those for Chapters 5, 6, 8 and 9, are listed in References – Part III, pages 267–290.

RESISTANCE TO VIRUS DISEASES

The Importance of Plant Virus Diseases

Symptoms of plant virus diseases have been recognized for many hundreds of years, although it has only recently been possible to identify and study the causal pathogens themselves. Good examples of typical symptoms of virus-induced flower breaking in tulips, now known to be caused by tulip mosaic virus, can be seen in many of the paintings of the seventeenth-century Dutch Masters. Virus diseases of the potato were introduced into Europe at least two hundred years ago from South and Central America, and from Europe they spread to many other parts of the world; these viruses have caused severe losses of yield ever since. Plant virus diseases are not, therefore, a modern problem, but most of the knowledge concerning plant viruses and the diseases that they cause has been gathered during the past two or three decades.

Some of the most damaging diseases of crop plants are caused by viruses, although it has been possible to assess this damage in detailed economic terms with only a very few diseases. The damage to the sugar beet crop caused by the virus yellows disease was estimated to be over £7 million in 1957 in the UK alone and in 1974 the comparable figure was more than £14 million. These losses represent a very significant proportion of the potential value of the entire crop. This disease has caused even greater losses in beet crops in many other parts of the world.

In some western parts of the USA, curly top disease has devastated sugar beet crops to such an extent in the past that sugar production from beet became uneconomic. Only after the introduction of curly top-resistant varieties and other control measures was it possible to resume beet growing on a large scale to the west of the Rocky Mountains.

Barley yellow dwarf virus is widespread throughout the temperate regions of the world and can seriously damage grass and cereal crops. Most of the other important agricultural and horticultural crop plants can be attacked by several viruses. Some crops, such as tomatoes, are susceptible to a large number of virus diseases, which not only decrease yields of produce but also affect the quality of the product.

Although many annual crops suffer very considerable losses from virus infections, perennial or vegetatively propagated crops are particularly vulnerable to virus diseases. For example, in the UK 'seed' potatoes are usually grown

in the more northern areas, including parts of Scotland and Northern Ireland, where the aphid carriers of many potato-infecting viruses are relatively few. Production of ware potatoes, i.e. those to be eaten, is centred in the more southern areas of the UK and the 'seed' tubers have therefore to be transported over a considerable distance. This, and other factors, raises the cost of 'seed' potatoes but it is necessary at present to avoid serious outbreaks of virus diseases which would be even more expensive to the ware grower.

Soft fruit (such as raspberries and strawberries) and top-fruit (such as apples and pears) are also often severely damaged by many viruses. In order to maintain economically acceptable levels of yield, it is often necessary to replace fruit bushes and trees at frequent intervals with virus-free propagating material. Tree crops, such as citrus and cocoa, are often badly attacked by virus diseases and infected trees have to be destroyed or replaced.

The causes of losses due to virus diseases are twofold. First, there are the losses which result directly from the effects of disease on the growth and yield of the host plant. Second, there are the costs of attempting to control the diseases, for example by applying pesticides to control the virus vectors, or of trying to avoid disease by planting with virus-free stock. Although these attempts rarely achieve a complete control of virus diseases, they help to ensure that losses are kept within economically acceptable limits. The use of resistant varieties is often the cheapest, and also sometimes the most effective, way of reducing damage by virus diseases.

Characteristics of Viruses and Virus Diseases

Plant viruses are submicroscopic entities which multiply only within living cells. Some viruses have such small or localized effects on their host plants that infection does not result in visible symptoms; these are called 'latent' viruses and cause a 'symptomless' disease in their host plants. It is important to realize, however, that viruses which are latent in one host may produce very severe symptoms in another.

Although several systems of nomenclature for plant viruses have been proposed, the system used in this book is the one in which viruses are named after the diseases which they cause: thus tobacco mosaic virus causes tobacco mosaic disease, potato leaf roll virus causes potato leaf roll and curly top virus causes curly top. For convenience, names are sometimes abbreviated to the initial letters of the complete virus name; for example, tobacco mosaic virus becomes TMV and curly top virus, CTV.

Most plant viruses consist of nucleic acid molecules surrounded by a protein coat. This nucleic acid is generally ribonucleic acid (RNA) but a few plant viruses contain deoxyribonucleic acid (DNA). The protein coat seems to have a protective role and is probably also important in determining how a particular virus is transmitted from one host plant to another. The protein coat is not essential because a few viruses, for example potato spindle tuber virus, consist only of nucleic acid (Sogo, Koller and Diener, 1973). The nucleic acid part of viruses is essential, because it carries the genetic information which instructs infected host cells to produce more viral nucleic acid and, usually, its associated protein. The production of new virus particles is therefore entirely at the expense of its host cell, which is usually damaged or weakened as a result.

Plant viruses are too small to be seen under the ordinary light microscope, but many different types of viruses have been identified under the transmission electron microscope at very high magnification. These viruses consist of individual particles of characteristic shapes and sizes. Those plant viruses visible under the electron microscope fall into four morphological categories: (1) those with 'spherical' or, more accurately, polyhedral particles. In such particles the subunits of the protein coat are arranged in icosahedral symmetry so that the particles have twenty identical faces. These symmetrical particles are often referred to as **isometric**. The protein coat is clearly visible under the electron microscope, but the nucleic acid cannot be seen because it is wrapped inside the protein; (2) those with rigid, rod-shaped particles e.g. tobacco mosaic virus. In these particles there is an inner 'tube' of nucleic acid surrounded by a 'tube' of protein subunits, and a coaxial hole which runs the length of the particle; (3) those with flexible rods, which are usually called 'filaments'. These have a structure and composition very similar to that of the rigid rod-shaped viruses. Examples of this group are beet yellows virus and potato virus X; (4) those with 'bullet-shaped' (bacilliform) particles, which have a clearly defined protein shell surrounding a layer of nucleic acid, which in turn encloses a hollow core. Sowthistle yellow vein virus, lettuce necrotic yellows virus and alfalfa mosaic virus are among the few plant viruses with this type of particle.

Virus particles which are rod-shaped, filamentous or bacilliform are asymmetric and are commonly referred to as **anisometric**.

Several viruses comprise particles of more than one size or shape. For example, tobacco rattle virus consists of two types of rigid rods with an identical diameter but of different lengths. Both types of particle must be present in a host cell for infection to occur. Alfalfa mosaic virus has at least four different types of particle, one of which is polyhedral and four bacilliform, all of which are necessary for infection. Each type of particle is essential if infection is to occur because it carries a different part of the genetic information.

It should be emphasized that the morphology of particles of many important plant viruses is not known because they have not been seen under the electron microscope.

Variability of Plant Viruses

The nucleic acid part of a virus initiates infection and carries the genetic code which gives the appropriate instructions to the host cell to reproduce or replicate viral nucleic acid. In TMV the RNA is present as a single strand or chain of about 6000 nucleotide molecules. There are only four types of nucleotide in the strand, each type having a different nucleotide base. The bases concerned are adenine, guanine, cytosine and uracil; these are present in different proportions in the nucleic acid strands of different viruses and sometimes in different strains of the same virus. For example, the 'M' strain of TMV has a higher proportion of pyrimidine nucleotides than other strains which have been examined. It is, however, the sequence in which the nucleotides are arranged in the strand which determines the genetic code of the virus. With 6000 nucleotides in a single TMV particle it can be seen that the combinations and permutations of the arrangement of the nucleotides are almost endless. If only one of these 6000 nucleotides is missing or out of place in the normal sequence, the genetic code is

changed and a new variant or virus strain is produced. It has been calculated that, in TMV, mutations occur naturally in living host cells once in 10^{11} virus particles. The possibilities for genetic variation through mutations in TMV, and presumably in other viruses also are, therefore, very great.

In viruses such as tobacco rattle virus, which consist of more than one kind of particle and therefore have a 'divided genome', another form of genetic variation is possible. There can be a reassortment of equivalent genes between the genomes of the different particle types. This makes possible an increased amount of variability over and above that resulting directly from the mutations themselves.

This potential genetic variability in viruses is of great importance to the plant breeder because of the danger that new resistance-breaking virus strains may arise. Most of the viruses that have been studied in detail are known to exist in a number of genetically distinct strains; nevertheless, genetic variability has been much less of a problem with viruses generally than with fungal or bacterial pathogens or insect pests. It will be seen from several examples in Chapter 9 that most of the virus-resistant varieties that have been developed so far have not been attacked by new resistance-breaking strains. There have, of course, been serious problems with virus strains in certain virus-host combinations, but these have been far fewer than would have been expected from experience with resistance to fungal diseases. The reason for this is not known, but it may be related to the very small number of genes, probably less than ten, that are present in most plant viruses. Most of the mutations that occur will therefore lead to the development of a number of allelic series at a few gene loci. This would presumably reduce the extent and range of genotypic variation that could occur in a particular virus.

Different strains of a virus can be identified in a number of ways, for example by comparing the type and severity of symptoms on a range of test plants, by serology, by cross-protection tests or by more sophisticated methods including immuno-electrophoresis. However, the distinctions between virus strains that are based on these methods are of very limited significance to the plant breeder, whose main interests concern the differential reactions between specific virus and host-plant genotypes and, in particular, resistance-breaking strains. No association has been found between the ability of virus strains to attack specific host genes and serological relationships. Some resistance-breaking strains may be less competitive than others, as with TMV in tomato.

Epidemiology and Transmission of Plant Virus Diseases

Before starting a programme of breeding for resistance to a particular virus disease it is desirable to know its epidemiology (i.e. the factors affecting the outbreak and spread). It is particularly important to know how the causal virus spreads in the field, what are its specific vectors, if any, and what are the relationships between the virus and its vector. It is also important to know whether there are alternative methods whereby the virus can be transmitted easily for use in screening and selection tests. Detailed accounts of ways in which viruses can spread from plant to plant may be found in text books on plant virology (e.g. Matthews, 1970; Gibbs and Harrison, 1976); some of the more important transmission methods are described very briefly below.

Plant viruses cannot penetrate the intact cuticle of a host plant unaided and they normally can be transmitted from one plant to another only with the help of a wound-causing agent, the vector. Plant viruses are, therefore, wound parasites. However, when one cell of a host plant has been infected by a virus it can usually multiply and spread from cell to cell, probably through the plasmodesmata, without further help.

Plant viruses are transmitted from one host individual to another by the following six methods:-

- (1) vegetative propagation and grafting;
- (2) dodder (*Cuscuta* spp.);
- (3) contact;
- (4) pollen and seed;
- (5) fungi;
- (6) invertebrate animals.

VEGETATIVE PROPAGATION AND GRAFTING

All viruses can be transmitted by grafting a part of an infected plant on to a healthy plant. Indeed, a few viruses have so far been transmitted only in this way. Grafting is used to propagate many important trees and bushes and, if the stock that is used for grafting is infected with a virus, it is usual for the scion also to become infected.

Many damaging virus diseases are spread by vegetative propagation involving the planting of diseased tubers, bulbs and cuttings. Most potato viruses are spread through the tubers because tubers from infected plants invariably give rise to diseased plants. It is therefore particularly important to use only virus-free propagating material in vegetatively propagated crops.

DODDER

Species of dodder, which are parasitic higher plants, are capable of acting as a 'transmission bridge' between virus-infected and healthy plants. As some dodders have a very wide host range it is sometimes possible to infect plants, using this bridge, with a virus which would not normally be able to attack them. This method of transmission, which can be considered as a special form of graft transmission, can be useful experimentally but is of no economic significance.

CONTACT

Many viruses can be transmitted by contact between healthy and infected plants, provided that the contact damages the tissues sufficiently to allow virus particles to be expressed from infected cells and for them to enter wounded cells of the previously healthy plant. If the wounding is too severe, however, no infection will occur. Contact transmission is very widely used in experimental work with viruses, although many of the most important viruses are not transmitted like this in the field. Viruses that can be transmitted by contact are often referred to as 'sap-transmissible' or 'mechanically transmissible'.

In the field, unintentional contact transmission is usually effected by touching delicate tissues of healthy plants with virus-contaminated hands, clothes or implements. Transmission can also occur by leaf-to-leaf contact within a crop when foliage is disturbed by wind or rain. Many economically important virus diseases are spread mainly by contact; for example TMV is often transmitted during cultural manipulations of the plants, such as removing the side shoots of tomatoes or tying plants to supports. If some of the plants are already infected with TMV, many healthy plants will become infected with virus transferred from those which are diseased.

Most viruses that are transmitted by contact are spread in other ways as well. For instance, potato virus X is transmitted from plant to plant in the soil by zoospores of a fungal pathogen *Spongospora subterranea*.

POLLEN AND SEED

Host-plant cells that are concerned with sexual reproduction, the egg and pollen mother cells, rarely become infected with viruses even in systemically infected plants. Some viruses, however, do infect the endosperm or embryo in the seed, and plants arising from infected seed are often themselves diseased. Examples of seed-transmitted viruses are bean mosaic virus in *Phaseolus* beans and lettuce mosaic virus in lettuce.

Transmission of viruses through the pollen is even more uncommon than is seed transmission. However, bean mosaic virus (which is also seed-transmitted) and barley stripe mosaic virus are spread in the pollen. Particles of these viruses in the pollen grains infect the stigma of healthy receptor plants, so that the egg cell becomes infected with these viruses and therefore produces infected seeds. In contrast to this situation, pollen transmission of raspberry bushy dwarf virus not only results in the production of infected seed but also in the infection of the seed-bearing parent. Beet cryptic virus is both seed- and pollen-transmitted in sugar beet (Kassanis, Russell and White, 1978).

FUNGI

Certain viruses are carried from plant to plant by particular species of soil-inhabiting fungi. These viruses are transmitted from root to root by motile zoospores of primitive fungi which parasitize the roots of crop plants. In some cases the virus particles adhere to the surface of the zoospores which are present in virus-infected roots. The zoospores can travel through the soil and attack the roots of other host plants. During this process the virus particles can enter some of the damaged host cells, and virus infection follows. Tobacco necrosis virus is transmitted in this manner by zoospores of the fungus *Olpidium brassicae*, which can parasitize the roots of a large number of crop species. Lettuce big vein virus is also transmitted by zoospores of this fungus, but in this case the virus is carried inside the spores.

INVERTEBRATE ANIMALS

More plant viruses are transmitted in the field by invertebrates, particularly Arthropoda, than by any other group of vectors. Insects belonging to the Order Hemiptera, which includes sap-sucking bugs such as aphids and leafhoppers,

are by far the most important vectors of plant viruses. These insects have mouthparts which are adapted for piercing plant tissues and sucking sap from them. There is usually a close and specific relationship between a particular insect vector and the viruses that it can transmit. For example, curly top virus is carried by the beet leafhopper *Cirulifer tenellus*, but not by the peach-potato aphid *Myzus persicae*. Conversely, the beet leafhopper cannot transmit the aphid-transmissible beet mild yellowing virus. The latter virus can be transmitted by *M. persicae* but not by the black bean aphid (*Aphis fabae*) although both insects feed on sugar beet, which is a host for this virus. The reasons for this vector specificity are not understood, but the nature of the protein coat of a virus may be important in determining the way in which it can be transmitted.

Some of the insect-transmitted viruses are acquired by an appropriate vector from an infected plant during very short feeding times, often of only a few seconds' duration. If viruliferous vectors are transferred to healthy host plants, they can usually inject the virus, within a few seconds, by probing into the epidermal cells of the host. Viruses that can be transmitted during such short feeding times do not usually persist in the vector for more than a few minutes, and are said to be **non-persistent** in the vector. Cucumber mosaic virus, potato virus Y and lettuce mosaic virus are examples of non-persistent aphid-transmitted viruses. Other viruses, for example potato leaf roll virus and beet mild yellowing virus, are acquired much more slowly from infected plants, often not until the vector has fed for two or three days: such viruses, however, usually persist in the vector for many days or even for the rest of its life, and are known as **persistent** viruses.

More than 160 agriculturally important viruses are transmitted by aphids, at least 60 of which can be transmitted by one species alone, *Myzus persicae*. These viruses include potato virus Y and potato leaf roll, beet yellows, beet mild yellowing and cucumber mosaic viruses. Several other Hemiptera, including whiteflies and mealy bugs, are important vectors of plant viruses.

Relatively few viruses are transmitted by insects belonging to Orders other than the Hemiptera. A few are spread by thrips (Thysanoptera), and some are transmitted by insects with biting mouthparts; for example turnip yellow mosaic virus (TYMV) is carried by beetles. These beetles can ingest virus-containing sap from infected plants and some of this is regurgitated while feeding on healthy plants. The mouthparts become contaminated with virus and transmission is effected.

Invertebrates other than insects have been shown to transmit several important plant viruses. Some species of mites (Arachnida) transmit ryegrass mosaic virus in the non-persistent manner; other mites transmit viruses, including wheat streak mosaic virus, in the persistent manner. Several species of nematodes (Nematoda), particularly those which are ectoparasites of roots, are vectors of many viruses, such as arabis mosaic virus and tobacco rattle virus.

It is important for a breeder to know how the causal virus of a disease is transmitted. He must know, not only the identity of any specific vector involved, but also the nature of the relationship between virus and vector. Only then can he begin effectively to plan how to conduct his screening and selection programme, whether in the field or in the glasshouse.

Types of Resistance to Virus Diseases

Many different types of resistance to virus diseases have been recognized.

Björling (1966) described six distinct types, classified into two main groups. The first group comprises (1) virus tolerance; (2) resistance to increase and spread of virus in an infected host plant; (3) resistance involving rapid death of infected tissues (hypersensitivity), resulting in the formation of chlorotic or necrotic local lesions at the inoculation site and (4) extreme resistance, or what is often wrongly referred to as 'immunity'. The second group is concerned with a tendency to escape disease and comprises (5) resistance to virus inoculation, and (6) resistance to the vectors.

A similar classification of types of resistance will be used in this book, although there are important differences between this system and that proposed by Björling (1966). The six types are as follows:

- (1) immunity;
- (2) resistance to virus infection;
- (3) resistance to establishment and spread of virus in host plant;
- (4) resistance to virus multiplication;
- (5) tolerance;
- (6) resistance to vectors.

IMMUNITY

Plants that are immune to a particular virus disease show no reaction whatsoever when inoculated with the virus, and the virus does not multiply in them. This definition of immunity is equally valid for inoculation by grafting, contact or vector. It is known that beet yellows virus (BYV) cannot infect barley, and that barley yellow dwarf virus (BYDV) cannot infect sugar beet. Thus barley is immune to BYV and sugar beet to BYDV. Immunity is absolute. There are no degrees of immunity. A plant is either immune to a particular virus or it is to some extent susceptible to it.

Most plants are immune to most viruses and susceptibility is therefore the exceptional condition. The reasons for this 'general' resistance of plants to viruses are not understood and more work is urgently needed to elucidate the nature of such resistance. Until we have this information it is difficult to assess its potential usefulness to the plant breeder. In the meantime, the fact that sugar beet and potatoes are immune to both barley yellow dwarf and cocoa swollen shoot virus is of very little practical interest or importance. Of greater immediate interest is the possibility that immunity to barley yellow dwarf virus may exist in a wild relative of cultivated barley, and that this character might be introduced into barley by interspecific hybridization.

It is very unlikely, however, that true immunity to a virus will be found within a crop species or its near relatives. Most, perhaps all, of the reports of 'immunity' to virus diseases refer to a type of resistance where the virus does infect host cells, but the infection is confined to one or a few cells near the site of inoculation. This 'extreme resistance' is apparently due to a very active response of the host plant to infection and is therefore the very antithesis of true immunity. In practice it can be very difficult to distinguish between immunity and extreme resistance without very detailed histological examination of cells in inoculated tissues. Nevertheless, it is important to realize that the two types of resistance are quite distinct, and that they involve different mechanisms.

Although immunity to viruses is of limited immediate practical use to the plant breeder, it may be possible eventually to introduce immunity to certain viruses into cultivated crop species from other species, genera or even families by techniques such as hybridizing cells of different organisms. If this could be done, barley breeders might become very interested in the fact that sugar beet and potatoes are immune to barley yellow dwarf virus. With present technology, however, breeding for immunity to most viruses is not a reasonable objective and the breeder must seek to exploit other kinds of resistance.

RESISTANCE TO VIRUS INFECTION

A heritable tendency not to become infected when exposed to infection with a virus to which it is susceptible, can be a very useful characteristic in a virus-resistant variety, because it slows down very significantly the rate of development of an epidemic in the field. This type of resistance has been very widely used, often in conjunction with other forms of resistance, in breeding for resistance to viruses in many crop species.

A tendency to escape infection has been reported with both contact and vector-transmitted viruses, although it is probable that quite different mechanisms of resistance may be involved. Troutman and Fulton (1958) reported that when tobacco leaves were contact-inoculated with cucumber mosaic virus (CMV) only about one-twentieth as many local lesions developed on leaves of a tobacco line TI 245 as on similarly inoculated leaves of several other varieties. This disease escape was also effective against four other contact-transmitted viruses including TMV and tobacco necrosis virus (TNV). Holmes (1961) showed that this resistance of TI 245 to these viruses, and to five additional viruses, is controlled either by a single gene or by a group of closely linked genes. This contrasts with the situation reported in spinach, where a monogenically controlled tendency to escape CMV infection in a breeding line, PI 179590, is effective against only certain strains of this one virus (Webb *et al.*, 1960).

Resistance to TMV infection has been found in some tomato varieties (Holmes, 1955), and also in certain related species of *Lycopersicon*; these two sources of resistance have been extensively used in breeding virus-resistant tomato varieties. Resistance to infection with potato virus Y, which can be transmitted both by contact and by aphids, has also been used successfully in breeding new varieties of potato, tobacco and pepper.

Although resistance to infection with many contact-transmitted viruses has been utilized in many crop species, very little is known about the mechanisms that are responsible for this resistance. The thickness of the cuticle on the leaves of the host plant, and the size and number of leaf hairs, may influence the efficiency of virus transmission, but there is little direct evidence that these factors are important. The resistance of tobacco line TI 245 could not be ascribed to a smaller number of leaf hairs per unit area, but was correlated with the number of ectodesmata in the outer walls of epidermal cells in leaves of different tobacco varieties (Thomas and Fulton, 1968a). Ectodesmata are probably the main entry points for contact-transmitted viruses, and their number and condition may determine the relative susceptibility of different lines. The number of virus receptors, either at the cell surface or within the cell, may also determine the ability of different varieties to escape virus infection (Atabekov, 1975).

Many of the mechanisms of resistance to infection with contact-transmitted viruses might be expected to operate also with non-persistent vector-aided viruses, where virus particles are usually introduced into the epidermal cells of leaves or stems. It is not surprising, therefore, that the resistance of tobacco TI 245 is as effective against aphid transmission of CMV as it is against contact transmission of this virus. Aphids apparently transmit non-persistent viruses, such as CMV, by introducing virus particles into the ectodesmata or plasmodesmata at the junctions of epidermal cells (Yoshii, 1966); presumably fewer transmissions occur in plants with fewer of these protoplasmic connections in the epidermis.

Viruses of the persistent type are normally injected into the sub-epidermal tissues, particularly the vascular bundles. For example, beet yellows and beet mild yellowing viruses are introduced into phloem cells of sugar beet leaves during prolonged feeding probes by the aphid vector, *Myzus persicae*. Curly top virus and potato leaf roll virus are also injected into the phloem by their respective leafhopper and aphid vectors. There have been several reports of varietal differences in resistance to infection with these viruses under natural infection conditions in the field. For instance, more plants have remained free of curly top in some sugar beet varieties than in others, even in the presence of large numbers of viruliferous leafhoppers. Similar differences in resistance to infection with beet yellows and beet mild yellowing viruses have been found between different sugar beet breeding lines, but resistance to the two viruses was not always shown by the same line (Russell, 1972a). A tendency to escape infection by leaf roll virus was first reported in potatoes in 1926, and this resistance has been used in potato breeding programmes. Several varieties have recently been marketed which have some resistance to potato leaf roll virus infection. For example, in 1975 the average percentage of leaf roll-infected plants in one series of trials in Scotland was 0.3 per cent in Pentland Crown compared with 16.8 and 15.8 per cent in the most susceptible varieties. Of early potato varieties, Ulster Sceptre had 1.0 per cent infected plants compared with 20.5 per cent in the most susceptible variety. Pentland Crown and Ulster Sceptre, therefore, show a pronounced tendency to escape infection with leaf roll virus. Similar advances in breeding for resistance to infection with this virus have been achieved in other countries.

RESISTANCE TO SPREAD OF VIRUS

In plants that show this kind of resistance, a virus remains localized and there is little, if any, spread of virus from the site of inoculation. Several different mechanisms can inhibit or delay the spread of a virus in a host plant. Although the inherited factors which affect the establishment and spread of viruses can operate independently, it is often impossible to dissociate these two stages of resistance; they are, therefore, considered together in this section.

The most common resistance mechanism affecting the establishment and localization of spread of viruses is **hypersensitivity**, where infected cells die prematurely. A very localized hypersensitive response to inoculation, often involving only one or two adjoining cells, is sometimes referred to as 'extreme resistance'. Groups of cells, which are damaged or killed as a result of hypersensitivity following contact inoculation with a particular virus, are visible

to the naked eye as chlorotic or necrotic spots, known as **local lesions**, on inoculated leaves. The causal virus normally remains in the local lesions or in the tissues immediately adjacent. However, necrosis of these cells does not always stop the spread of the virus, suggesting that cell necrosis and virus localization are separate aspects of hypersensitivity. In addition, virus localization can often be reversed, so that virus spread can be made to occur by altering the light intensity or the temperature in which inoculated plants are grown. Thus, leaves of *Nicotiana glutinosa* that have been contact-inoculated with TMV usually develop well-defined necrotic local lesions, but at high temperatures inoculated plants develop a lethal systemic necrosis.

With most viruses, infections become systemic in susceptible hosts and spread for considerable distances from the points of inoculation. In some resistant host plants, systemic virus spread is suppressed or delayed, perhaps because of physical barriers, such as necrotic vascular tissues, or because of antiviral factors produced in response to infection.

The mechanisms responsible for virus localization at, or near, the point of contact inoculation are probably distinct from those that limit virus spread throughout the host plant. This conclusion is supported by work carried out on the infection of *Claytonia (Montia) perfoliata* with beet yellows virus (Russell, 1963). When this virus (BYV) is contact-inoculated with the aid of an abrasive, discrete red local lesions are produced; the virus is entirely confined to these lesions. However, when *C. perfoliata* plants are inoculated with BYV using prolonged feeds by viruliferous *Myzus persicae* aphids, the virus becomes systemic in the host plant. Viruliferous aphids cannot transmit BYV during short feeding probes into the epidermis, and virus particles must be injected into the phloem if infection is to follow. It appears, therefore, that the hypersensitive response to BYV occurs in the epidermis but not in the phloem of *C. perfoliata*. In *C. perfoliata* plants that are systemically infected with BYV, red spots resembling local lesions develop on the older leaves, including those which were not inoculated by viruliferous aphids; BYV particles presumably spread from within the leaf tissues to some of the epidermal cells where they induce a hypersensitive response which results in local lesions.

The contention that hypersensitivity is usually restricted to the epidermal cells is supported by experiments with TMV in *Nicotiana glutinosa* (Yarwood, 1960). If TMV is contact-inoculated on to leaves of *N. glutinosa*, local lesions are formed, but if a healthy *N. glutinosa* plant is grafted on to a systemically infected tobacco plant, systemic TMV infection of the *N. glutinosa* plant occurs. This again, shows that there is usually no hypersensitive response to infection when the epidermis is 'by-passed' at inoculation. For this reason hypersensitivity, which localizes contact-inoculated viruses, and the limitation of systemic virus spread, will be considered in separate sections below.

Hypersensitivity to virus infections has been widely exploited in breeding programmes of many important agricultural and horticultural crops including tobacco, tomatoes, potatoes and *Phaseolus* beans. There are many practical advantages for the plant breeder in working with hypersensitivity. Inoculation procedures are usually simple, thus enabling large numbers of plants to be tested for resistance using contact inoculation. It is usually easy to identify and count local lesions, and degrees of resistance shown by different plants can easily be compared. Hypersensitivity is also controlled by a small number of major genes and can, therefore, readily be exploited in a breeding programme. The main

drawback of hypersensitivity is that it is often very strain-specific; breeders have frequently been troubled by resistance-breaking virus strains that are not controlled by this form of resistance. Such strains have occurred in tomatoes with spotted wilt virus, in potatoes with virus Y and in tobacco and pepper with TMV. It will be noted that these three examples involve plants of the Solanaceae. Although problems of virus-strain specificity have been encountered with plants of many families, such problems seem to be more common in certain families and species of crop plant than in others. This raises the possibility that problems with resistance-breaking virus strains may be characteristic, not only of certain viruses or resistance mechanisms, but also of particular taxonomic groups of host plants.

Mundry (1963) reported that strains of TMV that do not produce local lesions on *Nicotiana sylvestris* or Java tobacco, easily mutate to 'necrotic' strains which do produce such lesions. This observation confirms that the formation of local lesions does not depend on either the genotype of the host plant or of the virus alone, but depends on an interaction between certain genotypes of host and virus.

Very extensive investigations have been carried out on the biochemical and physiological nature of hypersensitivity to virus infections. It is generally agreed that the formation of virus-induced local lesions is associated with a rapid increase in the activity of certain enzymes, particularly polyphenol oxidase and peroxidase, in the affected tissues. Toxic quinones are commonly formed from dihydric phenolic compounds (Hampton and Fulton, 1959) and quinones are both virus-inhibitors and phytotoxic to the host cells. The death of an infected cell in a hypersensitive reaction may not, therefore, be the *cause* of resistance but, rather, a concurrent phenomenon related to the inhibition of the causal virus.

Factors other than an increase in phenolic and related compounds during local lesion formation have also been suggested to account for localization of virus infection. For example, Weintraub and Ragetli (1961) showed that calcium is laid down around TMV lesions in leaves of *Nicotiana tabacum* and *N. glutinosa*. This and other reactions suggests that infection with TMV may set off premature senescence of cells, the normal ageing process being telescoped into a few days, or even hours. If this is so, virus infection may cause instant ageing, so that infected cells die without supporting virus multiplication. It is interesting that increased rates of respiration, the evolution of ethylene and the development of colour changes are characteristic both of natural senescence of leaf tissues and of virus-induced local lesions.

In some varieties of tobacco, a zone about 1–2 mm in diameter surrounding each local TMV lesion develops a high level of resistance to TMV (Ross, 1961a). This 'localized induced resistance' is presumably caused by the outward diffusion of virus-inactivating compounds from a local lesion into the surrounding tissues; the nature of such compounds is uncertain although it has been shown that phenolic substances and certain carbohydrates accumulate in this resistant zone. Ross (1961a) showed that the zone surrounding TMV local lesions on tobacco leaves is resistant also to tobacco necrosis, tobacco ringspot and tomato ringspot viruses. However, in *Phaseolus* the zone surrounding TMV lesions is resistant to TMV but not to tobacco necrosis virus or alfalfa mosaic virus (Yarwood, 1960). It seems, therefore, that the specificity of the resistance in these zones is a property of the host plant rather than of the viruses that are involved.

Localization of virus infection by hypersensitivity concerns a number of interrelated and complicated processes, the nature of which is imperfectly understood. Nevertheless, hypersensitivity is one of the easiest and most effective types of resistance to viruses which is available to the plant breeder. It is unfortunate that its effectiveness in disease control has often been reduced by race-specificity.

The extent or rate of virus spread in systemically infected plants is often limited or delayed in resistant plants. This resistance to virus spread has, like hypersensitivity, been used very widely in many species of crop plants. For example, plants of certain potato varieties produce smaller percentages of virus-infected tubers than others when infected with potato virus Y. Beemster (1972) has shown that the systemic distribution of potato leaf roll virus and potato virus Y is slower in old than young potato plants, and systemic spread of viruses is also slower in some potato varieties than others. Cuttings, buds and shoots of certain varieties of sugar cane are often virus-free even when these are taken from plants which are systemically infected with sugar cane mosaic.

As with hypersensitivity, there are probably many different heritable factors which can affect the extent and rate of virus spread within a systemically infected host plant. Virus spread from cell to cell may be restricted in some way, perhaps by a blockage of plasmodesmata or by deposition of callose or other compounds, as suggested by Shimomura and Dijkstra (1975). Another possibility is that the translocation system in some host genotypes is less suitable, in some unexplained way, for the rapid transport of virus particles. Another is that virus infection may cause necrosis of cells in the translocation system, particularly the phloem, so that virus transport is inhibited. Such virus-induced phloem necrosis is known to occur in systemically infected plants as, for example, in sugar beet infected with beet yellows virus or curly top virus. In these particular examples, however, it is uncertain whether such reactions are effective in reducing spread of these viruses. There is also the possibility that some form of antiviral factor, either pre-existing in the host before infection or formed in response to infection, is responsible for limiting virus spread. Experimental work has confirmed that systemic antiviral factors do exist but their importance in breeding for resistance to virus diseases is still unknown.

Ross (1961b) found that when half leaves of plants of a tobacco variety, Samsun NN, were inoculated with TMV, the uninoculated half leaves and the upper leaves of the plant showed a high level of resistance to subsequent TMV inoculation. The uninoculated parts of the plants were found to be free of TMV particles and this resistance could not, therefore, be attributed to cross-protection mechanisms. The resistance, which was first detectable in uninoculated half leaves 2–3 days after inoculation and which persisted for more than 20 days, has been called 'systemic acquired' or 'induced' resistance.

Localized induced resistance in tissues surrounding local lesions usually gives complete resistance to the causative virus. In contrast, systemic induced resistance confers only partial resistance, local lesions being produced on resistant leaves, but these are fewer and smaller than on corresponding susceptible leaves.

There has been much conjecture concerning the nature of the antiviral factors (AVF) that are involved in localized and systemic induced resistance. Several workers have suggested that separate AVFs are involved in the two phenomena, and it is presumed that the AVF concerned in systemic induced resistance is more readily diffusible than the other. Bozarth and Ross (1962)

proposed that a metabolite, which is translocated from the site of infection, triggers off resistance mechanisms within virus-free cells and does not itself directly confer resistance. Sela, Harpaz and Birk (1965) found that the production and action of a systemic AVF was non-specific, induced resistance to one virus being effective also against several others. This AVF, which is probably a protein, has many properties in common with interferons (Atanasoff, 1963). Interferons are antiviral proteins or polypeptides which are produced in animal and plant cells in response to virus infection; they are rapidly liberated from infected cells and work extracellularly (Loebenstein, 1972). Four antiviral proteins have recently been identified in plants, production of three of which can be stimulated by injection with polyacrylic acid (Gianinazzi and Kassanis, 1974).

Antiviral factors that are associated with localized induced resistance have many similarities with **phytoalexins**, which are produced by higher plants in response to infection with fungi or other biological or physical stimuli. These are phenolic compounds which do not occur beyond the site of primary infections and which strongly inhibit the growth of certain micro-organisms (Cruickshank, 1963). It seems likely, therefore, that localized AVFs may also be phenolic compounds, resembling phytoalexins, and that AVFs concerned with limiting systemic virus spread are proteins or polypeptides.

This work on AVFs has several important implications. If two separate AVF systems are active in higher plants, it is obviously desirable that both should be employed in breeding for resistance to viruses that are spread mainly by contact inoculation. With viruses that are normally inoculated into subepidermal tissues, such as insect-transmitted viruses of the persistent type, which are injected into the vascular system of the host plant, localized induced resistance is of little use. However, high concentrations of systemically acting AVFs might restrict the spread of these viruses and reduce the virus concentration in the host plant. There might also be the added benefit of protecting the host plant against infections of other viruses. The development of resistant varieties with a high capacity to generate AVFs of any kind, when challenged by a virus, would be highly desirable. Chadha and MacNeill (1969) have shown that certain tomato varieties have the ability to produce high concentrations of AVFs in plants that are systemically infected with TMV. It is important for virologists and plant breeders to study the question of AVFs in more detail with the aim of developing simple but reliable tests which could be used on a large scale in breeding for resistance to virus spread.

RESISTANCE TO VIRUS MULTIPLICATION

Viruses can readily infect host plants that show this kind of resistance, but they do not multiply in such hosts to the same extent, or as rapidly, as in more susceptible plants. This resistance to virus multiplication is similar in many ways to resistance to systemic virus spread; some of the same mechanisms may be involved in both types of resistance. An AVF, which was formed in tomato plants that were systemically infected with TMV, decreased the concentration of virus in the plant in addition to restricting virus spread (Chadha and MacNeill, 1969).

Several allelic genes in barley are known to control resistance mechanisms that limit the multiplication of barley yellow dwarf virus (BYDV). Multiplication of BYDV is greatly reduced in resistant plants which are growing vigorously, but is much less reduced in slowly growing resistant plants which are infected with a 'virulent' virus isolate. This shows that the expression of resistance to BYDV multiplication in barley depends on several factors, including the virus strain and host genotype involved, and the vigour of the host plant. Resistance is partially dominant in certain environments, particularly those which favour rapid growth of the host plants, and is recessive in others. These same alleles apparently also decrease the severity of symptoms on the leaves, and also the loss of grain yield resulting from virus infection, presumably because of the lower virus concentration in resistant plants. This resistance is, therefore, difficult to distinguish from virus tolerance. Resistance to virus multiplication is also associated with virus tolerance in several other virus-host combinations, but with beet yellows virus and sugar beet, tolerant plants sometimes contain even higher concentrations of virus than do sensitive plants (Russell, 1966d).

There are many other examples of the successful exploitation of resistance to virus multiplication. The Ambalema variety of tobacco has a high level of resistance to TMV multiplication (Bancroft and Pound, 1954). Reduced virus concentrations have been found in varieties of spinach that are resistant to cucumber mosaic virus (Pound and Cheo, 1952), and in lettuce that is resistant to lettuce mosaic virus (Tomlinson and Faithfull, 1975). Multiplication of cucumber mosaic virus is also suppressed in certain cucumber varieties, this resistance being controlled by a single dominant gene (Wasuwat and Walker, 1961).

The mechanisms that are responsible for resistance to virus multiplication are not understood, but several resistance mechanisms are probably involved. The genome of a virus is fully expressed only if the protein coat is removed in an infected host cell; any factors in the cell which interfere with the complete removal of the protein coat could inhibit virus replication. Alternatively, virus replication could be suppressed by factors which affect the formation or activity of viral RNA. If the correct assembly of intact virus particles from their component parts were prevented in some way, virus multiplication would be greatly inhibited.

Evidence concerning the importance of such 'passive' resistance mechanisms is, at present, entirely lacking because of the technical difficulties involved in studying them. There is, however, some evidence that 'active' or 'responsive' resistance mechanisms, which operate as a result of virus infection, are at least partly responsible for decreased multiplication of CMV in resistant cucumber (Barbara and Wood, 1974). Nevertheless, Pound and Cheo (1952) were unable to correlate resistance with the concentration of virus inhibitors in the sap of different cucumber varieties.

Catherall and Hayes (1966) have reported that aphids can acquire and transmit barley yellow dwarf virus (BYDV) more effectively from certain barley varieties than from others. This suggests that resistance to virus multiplication can also occur with persistent, aphid-transmitted viruses in addition to contact-transmitted viruses.

Although many complex and imperfectly understood mechanisms are responsible for resistance to virus multiplication, this type of resistance has been exploited in many crop species. This emphasizes that successful programmes of

breeding for resistance can be carried out without understanding the underlying resistance mechanisms which are involved. It is comparatively easy to compare the concentrations of many viruses in different plants, for example by serology or quantitative local lesion assays using appropriate test plants, or by aphid transmission tests such as those used by Catherall and Hayes (1966). Plant breeders should be encouraged to use resistance to virus multiplication whenever possible. In combination with other types of resistance, this resistance should greatly help to reduce or delay spread of virus diseases in the field.

VIRUS TOLERANCE

The term 'virus tolerance' has been used in a number of different ways and this has, understandably, led to considerable confusion among plant breeders and virologists. The confusion seems to have arisen largely because there are at least three different kinds of tolerance (*Table 8.1*).

The first kind concerns plants in which a virus is able to multiply, but in which symptoms do not appear. In breeding for resistance to virus yellows of sugar beet, many such plants have been encountered but, in spite of the absence of visible symptoms on the leaves, these plants frequently suffered significant losses of root or sugar yield when infected (Russell, 1964a). A Japanese cucumber variety, Natsufushinari, showed no symptoms when infected with cucumber mottle virus but lost as much of its potential yield, in terms of numbers of fruits per plant, as varieties which showed severe symptoms (Kooistra, 1968). In this book, plants which show the type of virus tolerance in which virus infection damages the host but does not produce symptoms will be called **symptomless carriers**.

The second main kind of tolerance concerns plants which develop disease symptoms which are as severe as those in other plants, but which suffer less damage in agricultural terms; this is sometimes referred to as **disease tolerance**. Many breeding lines of sugar beet show severe yellowing symptoms on the leaves when infected with virus yellows, but nevertheless yield large roots with a high sugar content.

Table 8.1 DIFFERENCES BETWEEN VARIOUS TYPES OF VIRUS TOLERANCE

		<i>Amount of economic damage caused by virus infection (eg reduction of yield)</i>	
		<i>Severe</i>	<i>Slight</i>
<i>Intensity of virus symptoms on host (eg leaf discoloration)</i>	<i>Severe</i>	Sensitivity (intolerance)	Disease tolerance
	<i>Slight</i>	Symptomless carrier	True tolerance

In a third type of tolerance (**true tolerance**), virus-infected plants do not show severe disease symptoms and they are less damaged by infection than other

plants. For example, infected barley plants that are tolerant to barley yellow dwarf virus do not show pronounced yellowing or stunting of the leaves, and they give an acceptable yield of grain in spite of being infected.

Although these three types of virus tolerance differ in important aspects and have different epidemiological implications, they have one thing in common: the virus concentration is not necessarily reduced in any type, although virus tolerance and resistance to virus multiplication are often associated. Plants which react to virus infection with severe symptoms and significant damage are referred to as **sensitive** or **intolerant**.

Apart from cosmetic effects there seems generally to be little justification in breeding only, or mainly, for the lack of visual symptoms (i.e. to select symptomless carriers). However, an absence of visual symptoms is sometimes far more important even than the amount of yield reduction that is caused by disease. In salad crops such as lettuce, yield is of secondary importance to appearance, because a plant which shows severe yellowing of the leaves as a result of virus infection is unacceptable to the consumer and is therefore worthless. Plants of 'symptomless carrier' varieties might be acceptable even if they were made smaller by virus infection. In such cases, therefore, even the deliberate selection for the 'symptomless carrier' type of tolerance might be justified.

Many virologists and plant breeders have been very critical of the use of virus tolerance in breeding programmes although it has been, perhaps, the most widely used kind of resistance in the past. Bawden (1964) has emphasized the potential dangers of using virus-tolerant crop varieties because they might become important sources of infection for other, less tolerant varieties or for other susceptible crop species. Varieties that are symptomless carriers can obscure the need for other control measures and can make control by roguing almost impossible. Nevertheless, the dangers and difficulties of using virus tolerance should not be overemphasized. In the USA, virus-tolerant sugar beet varieties have been blamed for outbreaks of curly top on tomato crops, by acting as a gigantic virus reservoir of the curly top virus. There is no reason to believe, however, that tolerant varieties of sugar beet are more of a nuisance in this respect than sensitive varieties would be – it is merely that only curly top-tolerant varieties of beet can be grown in that region. Tolerant plants are no more likely to become infected than other plants, nor is the virus concentration always higher (or lower) in tolerant plants.

Even in perennial and vegetatively propagated crops, tolerance has given apparently trouble-free benefits over long periods. Posnette, (1969) cites tristeza virus in citrus and several viruses of pome fruits as two of many examples of successful tolerance breeding. Sugar cane mosaic is no longer an important disease in the USA because of the widespread use of virus-resistant varieties, in which virus tolerance is an important factor (Summers, Brandes and Rands, 1948). Nevertheless, in perennial and vegetatively propagated crops it may be advisable to use virus-tolerant varieties, particularly those which are symptomless carriers, only where other control methods are too expensive or ineffective, or as an insurance against failure of other methods.

Virus tolerance is simply inherited in many virus–host combinations. For example, tolerance to barley yellow dwarf virus is controlled by several major genes, some of which are reported to be allelic. An incompletely dominant gene *Yd*₂ confers a high level of tolerance and a recessive gene *Yd*₁, a lower level. Tolerance to bean yellow mosaic virus is controlled by two or three major genes

in *Phaseolus* (Baggett, 1956) and by a single dominant gene in red clover (Diachun and Hensen, 1959).

In other virus–host combinations, however, virus tolerance is controlled by polygenes. Sugar beet populations, derived from paired crosses between virus yellows-tolerant plants and sensitive plants, showed a continuous variation in level of tolerance, ranging from very tolerant to very sensitive; tolerance to sugar beet virus yellows is, therefore, controlled by many genes, as is tolerance to curly top virus in sugar beet. There is no general rule concerning the number of genes that are involved in the inheritance of virus tolerance.

Resistance-breaking virus strains have not been a major problem with most virus-tolerant varieties. However, virus tolerance is not always non-race-specific. For example, certain Ethiopian barleys are tolerant to barley yellow dwarf virus in some parts of the USA but not in others, and interactions between certain virus strains and host genotypes have been demonstrated experimentally (Gill and Buchannon, 1972). Plant breeders should, therefore, test their virus-tolerant breeding material against as wide a range of virus strains as possible.

Selection for improved levels of virus tolerance can be effected in various ways, depending on the types of tolerance involved. Wherever possible, selection tests should be carried out in the field in conditions where natural infection can be expected. There are two main advantages in using natural infection. First, there is a saving in time and trouble for the breeder who does not need to induce an artificial epidemic. Second, artificial epidemics may, in some cases, provide spurious or misleading results. For example, if the inoculum intensity is too high, small but significant differences in tolerance between lines or varieties may be masked. In selecting for resistance to curly top virus in sugar beet, where tolerance is an important component, breeding lines are planted in fields adjacent to the main breeding grounds of the leafhopper vector (*see* page 244). Every plant in a selection field therefore stands an excellent chance of becoming infected with curly top, and plants or lines which grow well in spite of infection can easily be selected for subsequent breeding.

Selection for tolerance to barley yellow dwarf virus (BYDV) in barley has often been carried out in the field, under conditions of natural virus spread, in many parts of the world. Plants have also been inoculated in field or glass-house tests by infesting them with viruliferous aphid vectors. The severity of symptoms on the leaves and the degree of stunting caused by infection can then be assessed and the least affected plants are selected (Catherall and Hayes, 1966). In such tests it is not always necessary to compare the yields of grain given by BYDV-infected and healthy plants of each genotype, in assessing tolerance, because loss of grain yield is closely correlated with the amount of virus-induced stunting and severity of yellowing symptoms on the leaves.

In selecting for tolerance to curly top virus (CTV) in beans (*Phaseolus vulgaris*) and tomatoes, rows of breeding lines are interplanted with rows of infected sugar beet plants on which the leafhopper vectors of the virus are encouraged to breed (Thomas and Martin, 1969). In this way a uniform spread of CTV from sugar beet to the rows of beans or tomatoes can be obtained. Very sensitive genotypes are killed or very severely stunted by CTV and selection of the most tolerant genotypes can therefore easily be carried out. The results of these very successful programmes of selecting for resistance to CTV in dwarf beans are shown in *Figure 8.1*. This resistance to CTV appears to incorporate resistance to virus infection in addition to virus tolerance.

In breeding for tolerance to virus yellows of sugar beet, more sophisticated selection techniques have been employed. It has been necessary to assess the effects of virus infection on the size and sugar content of the root in order to select tolerant plants effectively (Russell, 1964a). The amount of stunting of the foliage and the severity of leaf yellowing is not a reliable guide to the amount of root damage. As sugar is extracted only from the roots, the severity of symptoms on the leaves is, therefore, only of secondary importance.



Figure 8.1 A demonstration of resistance to curly top virus in dwarf beans Phaseolus vulgaris at the USDA Research Station, Prosser, Washington, USA. A row of a resistant variety is flanked by rows of susceptible varieties that have died as a result of infection.

Differences in virus tolerance between host genotypes are usually quantitative rather than qualitative, and are therefore less clear-cut than are differences in some other types of resistance. Although plant breeders prefer to deal with qualitative characters in their breeding material they have, nevertheless, been able to make very significant advances in breeding for tolerance to viruses in many crops.

RESISTANCE TO VECTORS

Plants that are completely resistant to the vectors of a particular virus should, accordingly, remain free of virus infection. It is not surprising, therefore, that much work has been carried out in breeding for resistance to the vectors of many important virus diseases. This effort has been concentrated mainly on insect vectors, although some work has been carried out with mites. The possibilities of resistance to ectoparasitic nematode or fungus vectors of viruses seem not to have been investigated on any large scale. Although resistance of crop plants to insects and mites is discussed in some detail in Chapter 10, it will also be considered briefly here insofar as it affects the epidemiology of virus diseases.

As pointed out earlier (*see* page 215) two types of virus, non-persistent and persistent, can be distinguished when considering vector transmission. Painter (1951) recognized three main categories of plant resistance to animal pests: non-preference, antibiosis and tolerance (*see* page 306). Any type of resistance which encourages a vector to feed briefly on one host plant and then to move immediately to other plants is likely to increase the spread of non-persistent viruses in a crop. For example *Myzus persicae*, an aphid vector of sugar beet mosaic virus (SBMV), which is a non-persistent virus, might feed only briefly on a non-preferred virus-infected sugar beet plant, but would nevertheless acquire the virus. This aphid could then transmit SBMV to adjacent, healthy beet plants during a search for a more-preferred host. However, there would probably be fewer vectors to transmit viruses in a crop of non-preferred plants and this would offset, to some extent, the potential increase in virus spread which would follow the more frequent aphid movements between plants.

Antibiosis, which inhibits the growth and multiplication of vectors on resistant plants, will also reduce the numbers of vectors that are present in a crop to spread viruses. This reduction in vector population should, therefore, indirectly decrease the spread of viruses in vector-resistant crops, particularly where most of the virus spread is within the crop itself and not from outside the crop.

Some of the likely effects of different types of resistance to vectors on the spread of persistent and non-persistent viruses in the field are summarized in *Table 8.2*. The effects of different types of vector resistance on the spread of semi-persistent viruses (e.g. beet yellows virus), which have intermediate transmission properties, would be similar to the effects on the spread of persistent viruses. It is unlikely that non-preference resistance would effectively control

Table 8.2 PREDICTED EFFECTS OF NON-PREFERENCE AND ANTIBIOSIS TYPES OF RESISTANCE TO VIRUS VECTORS ON VIRUS SPREAD AND VECTOR POPULATIONS

<i>Type of host-plant resistance to vector</i>	<i>Direct effects on virus spread</i>		<i>Effects on vector populations</i>
	<i>Non-persistent viruses</i>	<i>Persistent viruses</i>	
Non-preference (non-acceptance)	Possibly increased	Decreased	Decreased*
Antibiosis	No direct effect	No direct effect	Decreased*
Tolerance	None	None	None

*Decreased vector populations should indirectly decrease virus spread between plants within a crop but would not affect spread of virus *into* a crop.

the spread of non-persistent viruses, but it should reduce the spread of persistent viruses. Antibiosis would not directly control the spread of either type of virus, although it would do so indirectly by reducing the overall population of vectors. It is important for the plant breeder to decide which type of vector resistance is likely to be of most benefit in controlling a particular virus disease, so that he can plan his selection and testing programme accordingly.

Selection and Breeding Methods

Where one type of resistance is very effective on its own against a virus disease, particularly if this resistance is controlled by major genes, plant breeders can usually devise simple and effective ways of screening large numbers of plants for resistance. For this reason, simply inherited forms of resistance, such as hypersensitivity, have been preferred by breeders. A possible danger of such resistance, however, is that it may soon be overcome by a resistance-breaking strain. As with fungal and bacterial diseases, the advantages of the more easily used forms of resistance may often be offset by their transient nature. Also, as with breeding for resistance to diseases caused by fungi, resistance to virus diseases will depend on the breeding system of the crop species, the nature of the viruses, particularly their transmission characteristics, and the sources of resistance available.

References

The references cited in this chapter, together with those for Chapters 5, 6, 7 and 9, are listed in References — Part III, pages 267–290.

EXAMPLES OF RESISTANCE TO VIRUS DISEASES

Potatoes

Potatoes are propagated vegetatively by tubers through which many virus diseases are transmitted from generation to generation. Plants that become systemically infected with a virus (primary infection) generally produce tubers that are also infected, and these usually give rise in turn to virus-infected plants (secondary infection). Virus diseases have caused severe losses of yield in Europe and North America for at least 200 years but it is only in the past two or three decades that the causal viruses have been identified and characterized. The three viruses which are of particular interest and importance are potato leaf roll virus, potato virus X and potato virus Y. Progress in breeding for each of these viruses is briefly summarized below.

POTATO LEAF ROLL VIRUS

Potato leaf roll is one of the most damaging diseases of potatoes and is a frequent cause of degeneration of seed stocks in areas where its aphid vectors are common (*see Figure 9.1*). Treatment of potato crops with insecticides, applied either as foliar sprays or as granules to the soil, can sometimes delay the spread of leaf roll, but a more effective control measure has been to grow 'seed potatoes' (i.e. tubers for propagation) in areas where aphids are not usually numerous. In Britain, for example, seed potatoes are mostly grown in parts of Scotland, Northern Ireland and northern England where the climate is not conducive to the build-up of large aphid populations. For many years this measure, supplemented with insecticidal control of aphids where necessary, gave such a good control of potato leaf roll and potato virus Y in the UK that breeding for resistance to these viruses was often considered to be of much less importance than many other breeding objectives. However, aphid vectors of potato viruses became very numerous in many seed-producing areas in 1975 and 1976, and this was followed by a marked increase in the proportion of virus-infected seed tubers. This increased aphid attack was associated partly with a series of mild winters and warm summers, partly with the development of insecticide-resistant populations of *Myzus persicae*, the principal vector of these viruses, and partly with a change in economic circumstances which led to some

seed growers allowing crops to grow on to produce both seed and ware potatoes. This new situation thus emphasized the importance of developing virus-resistant and aphid-resistant varieties of potatoes.

Programmes of breeding for resistance to leaf roll have been carried out in many parts of the world including the UK (Howard, 1970; Davidson, 1973), the Netherlands (Wiersema, 1972), New Zealand (Bedi, 1975), the USA (Mackinnon, 1970; Davies, McEwan and Dixon, 1975), West Germany (Baerecke, 1961), Eastern Europe (Zadina, 1974; Gáspár and Bukai, 1975) and the USSR (Kameraz *et al.*, 1974; Kvasnikova, 1976). 'Extreme resistance', often referred to as 'immunity' in relation to virus diseases of potatoes, and hypersensitivity to leaf



Figure 9.1 Potato varieties differ in resistance to potato leaf roll virus. This photograph shows a plant of a susceptible variety that is prone to become infected and shows severe symptoms. Some varieties show a tendency to escape infection and others are tolerant. (By courtesy of Plant Breeding Institute, Cambridge)

roll are unknown, but several other types of resistance have been identified and used in breeding programmes. The main type of resistance used has been resistance to infection, which is expressed as an inherited tendency to escape infection (Mackinnon, 1970). Inherited differences in susceptibility to *Myzus persicae*, the principal vector of leaf roll, probably play an important part in resistance to infection. For example, high proportions of seedlings from several crosses between different potato clones were resistant to leaf roll when *Myzus persicae* was used as vector, and some of these were found to be resistant to the

feeding of *M. persicae* (Gáspár and Bukai, 1975). In New Zealand, the leaves of some varieties, including Whithu, were strongly resistant to leaf roll virus inoculation and these varieties suffered little yield reduction after inoculation (Bedi, 1975). Another variety, Rua, although very susceptible to inoculation, suffered little tuber loss, and can therefore be classified as tolerant. In most varieties, a high percentage of tubers from leaf roll-infected plants carry the virus, but there is usually little secondary infection in some varieties, including Pentland Crown and Pentland Dell. This tendency for leaf roll virus not to be transmitted through the tuber has also been observed in the UK, where Pentland Crown is recognized as being fairly resistant to leaf roll and very resistant to virus Y (National Institute of Agricultural Botany, 1977).

Although tolerance to leaf roll virus is known to occur widely in potatoes, plant breeders have tended not to select consciously for tolerance, because this type of resistance is usually considered to be dangerous in potatoes. It is feared that the stringent measures that are necessary to control spread of leaf roll and other aphid-transmitted viruses in virus-sensitive varieties, would not be applied as rigorously in tolerant varieties, the yield of which would be affected relatively little by virus infection. In particular, growers might plant infected tubers instead of those grown in areas where leaf roll is not common. In this way, tolerant varieties might become an important reservoir of leaf roll virus, and serve as infection foci for sensitive varieties. It is suggested that tolerance should therefore be avoided in vegetatively propagated crops such as potatoes, particularly when it is not difficult to breed for other, more desirable, forms of resistance. The replacement of all sensitive varieties with tolerant varieties would be justified only where virus-free seed stock is not easily available, or where infestations of viruliferous aphids are so heavy that other control measures, including other forms of resistance, give only a poor control of leaf roll.

In testing for resistance to leaf roll infection in the field, virus-free tubers of the clones to be tested are usually planted between rows of tubers that are known to be infected with the virus (Baerecke, 1961; Wiersema, 1972). Such tests must be carried out in areas where populations of *Myzus persicae* will develop so that spread of the virus will take place. Samples of tubers are harvested from the test plants of the clones and are planted in the following year to determine the frequency of infection. The proportion of virus-infected tubers can also be estimated using a histological test (the 'Igel Large' method) but this is not as reliable as identifying the proportion of diseased plants that develop from the tubers (De Bokx, 1972).

Although most selection for resistance to leaf roll has occurred under field conditions, Mackinnon (1969) showed that glasshouse tests can be used for detecting resistance. He reported that 41 out of 717 clones in a series of glasshouse tests remained free from leaf roll virus after repeated attempts to infect them using viruliferous *Myzus persicae*. Such glasshouse tests may prove to be a useful adjunct to field tests.

Sources of resistance to infection with leaf roll virus have been found in varieties of both Tuberosum and Andigena potatoes, and also in wild species such as *Solanum demissum*, *S. acaule*, *S. chacoense* and *S. stoloniferum* (Ross, 1966). *Solanum spegazzinii*, *S. microdontum* and *S. brachycarpum* have also been claimed to have good field resistance to leaf roll (Gerasenkova, 1974). Kameraz *et al.* (1974) have reported a complete absence of leaf roll infections

in progenies of *S. catarrhrum* × *S. tuberosum* crosses under conditions where other *Solanum* species and interspecific crosses became infected.

Very little is known about the genetics of resistance to infection with leaf roll virus, but this type of resistance is probably controlled by polygenes. Work by Baerecke (1958) and others has shown that crosses between moderately resistant plants give progenies that are often more resistant than either parent, indicating that there is transgressive segregation. The progenies of crosses between resistant and susceptible plants usually have an intermediate degree of resistance.

The mechanisms involved in resistance to virus infection are also imperfectly understood. As mentioned earlier, varieties differ in their resistance to the aphid vectors, and this may lead to a decrease or delay in the spread of virus in field crops; certainly, resistance to virus infection is much more effective in controlling spread of leaf roll when aphids are few. Necrosis of the phloem tissues occurs in some plants when infected with leaf roll virus, thus possibly restricting the spread of the virus within these plants (Hutton, 1949). McMillan, Mink and Locke (1969) showed that potato varieties differ in their ability to oxidize chlorogenic acid and that there is a negative correlation between the concentration of chlorogenic acid and resistance to leaf roll. As a potato plant ages, leaf roll virus is translocated from an inoculated leaf more slowly, until a stage of maturity is reached when almost no virus passes from an inoculated leaf to the rest of the plant (Beemster, 1972). This 'mature-plant resistance' may be an important factor in field resistance to leaf roll virus.

POTATO VIRUS Y

Potato virus Y (PVY) causes a mosaic disease in potato leaves and can lead to considerable loss of yield, the yields of individual plants being reduced by more than 50 per cent. The virus can be transmitted by sap inoculation, but most spread of PVY in the field is by aphids, particularly by *Myzus persicae*, in which it is stylet-borne and non-persistent. Some varieties are symptomless carriers of the virus but others are so sensitive to PVY that infection causes severe necrosis of the leaves. In most varieties, however, mosaic symptoms develop on the leaves of infected plants and wrinkling of the surface of the lamina (rugose mosaic) is also common.

In addition to these differences in tolerance, varietal differences have been reported in resistance to infection with PVY, hypersensitivity and 'extreme resistance' (or 'immunity'). Although tolerance to PVY has been demonstrated, virus tolerance is considered by most plant breeders to be an undesirable feature in potato varieties, because masking of visual symptoms might jeopardize other control measures. Breeding work has, therefore, been concentrated largely on selecting for resistance to infection, hypersensitivity and extreme resistance.

Good resistance to PVY infection is present in some potato varieties and also in *Solanum demissum*, *S. stoloniferum* and some other wild species. This resistance is apparently controlled polygenically, but little is known about the mechanisms involved. Methods of testing and selecting for resistance in the field are similar to those already described for potato leaf roll. However, the evaluation of resistance to PVY is more difficult than with leaf roll, because

plants that are grown from PVY-infected tubers do not always show distinctive symptoms; serological assays or tests with indicator plants, involving sap inoculation, are sometimes necessary. Hypersensitivity and extreme resistance are easier characters for plant breeders to use, and have therefore been utilized more often than resistance to infection, in developing virus-resistant varieties.

In hypersensitive plants the cells are so susceptible that infected tissues and areas surrounding them die quickly, thus restricting the spread of the virus to healthy tissues. Such plants, which are sometimes referred to as 'field immune', do not become systemically infected in the field. Although no variety is hypersensitive to all the most common strains of PVY, several varieties, including Epicure, King Edward and Majestic, are hypersensitive to the so-called 'potato virus C' which is an aberrant strain of PVY (Cockerham, 1943). The growing points and leaves of hypersensitive plants that have been infected by grafting, become necrotic and the whole plant eventually dies. This 'top necrosis' response, which is used as a diagnostic test for hypersensitivity to potato virus C in potato breeding programmes, is controlled by a single dominant gene, designated *Nc* (Howard and Fuller, 1965; Cockerham, 1970). Hypersensitivity to PVY is an example of race-specific resistance because it is effective against only some virus strains. For this reason, extreme resistance to PVY is used by plant breeders more extensively than hypersensitivity.

Extreme resistance to PVY, sometimes referred to as 'comprehensive resistance', is apparently effective against all known strains of the virus, whether the resistant plants are inoculated by sap, aphids or grafting. This non-race-specific resistance has been found in Neo-Tuberosum forms (Glendinning, 1975) and in several wild *Solanum* species including *S. stoloniferum*; this Mexican species has been very widely used as a source of this and other forms of resistance. Extreme resistance is simply inherited and plants that are resistant to PVY are resistant also to the related potato virus A. A simple but effective method of screening very large numbers of seedlings for extreme resistance to PVY has been described by Wiersema (1972). Seedlings are sprayed with inoculum containing PVY and carborundum by means of a spraygun; plants which show virus symptoms after three weeks are discarded and the remainder are retained for further testing. Alternatively, more conventional methods of sap inoculation can be employed, but these are more time-consuming.

At least three alleles are involved in controlling the resistance of *S. stoloniferum* to PVY (Cockerham, 1970). These, in decreasing order of dominance (Ross, 1958), are as follows: (1) *Ry*, which controls extreme resistance. No virus can be detected in the leaves or tubers of plants carrying this allele, even after graft inoculation (Delhey, 1975); (2) *Ryn*, which controls hypersensitivity; (3) *ry*, which confers susceptibility to infection and some degree of virus tolerance. An additional gene, *Rym*, is also probably implicated. The Tuberosum varieties, Bison and Fanal, carry the *Ry* gene in the simplex form (*Ry ry ry ry*) and Zadina (1977) suggests that these varieties may be a better source of resistance to PVY than *S. stoloniferum* because of their superior tuber quality and lack of undesirable features. Cockerham (1970) identified 12 genes which are concerned with resistance to PVY in various species of *Solanum*, including *Natbr* and *Nctbr* from *S. tuberosum* which control hypersensitivity to potato viruses A and C (strains of PVY) respectively, *Ny_{dms}* in *S. demissum* and six genes from *S. stoloniferum*. A single dominant gene controlling 'immunity' to PVY has also recently been recognized in some Andigena clones (Munoz, Plaisten

and Thurston, 1975). Plants carrying this gene are resistant to both sap and aphid inoculation, and no virus can be recovered from inoculated plants.

Plant breeders are fortunate in having several quite different types of resistance to PVY available. With the exception of virus tolerance, each type of resistance may, in future, play a part in the control of PVY by resistant varieties. Although some current varieties express resistance to infection, which can give a satisfactory control of PVY in the field, breeders have tended to concentrate more on breeding for hypersensitivity and extreme resistance. The main drawback of hypersensitivity is that it is not effective against all virus strains. Extreme resistance, which is effective against all strains, is simply inherited and is an easy character to use in programmes of selecting and testing. For these reasons, it appears that extreme resistance is likely to be the most useful type of resistance to incorporate in resistant varieties.

POTATO VIRUS X

Potato virus X (PVX) has a world-wide distribution and is probably the most common of all viruses in potatoes. It causes a disease which is often termed 'mild mosaic' and, as this term implies, the most usual symptom is an indistinct interveinal mottling on the leaves. These mosaic symptoms are so mild on plants of some varieties that they are scarcely visible; such tolerant varieties can act as important sources of infection for varieties which are more sensitive to PVX. Although the symptoms on the leaves are not generally severe, tubers from infected plants are fewer and smaller than those from virus-free plants, and some strains of PVX can cause yield losses of more than 50 per cent in very susceptible varieties. PVX is easily sap-transmissible and is spread in the field mainly by contact between healthy and diseased plants, and by means of virus-contaminated farm implements or clothing. Aphids do not transmit PVX, but the virus can be transmitted to roots of healthy plants by zoospores of the wart disease fungus, *Synchytrium endobioticum*, in the soil. Synergism between PVX and potato viruses A and Y can occur, and mixed infections of these viruses can reduce yields of tubers to a greater extent than would be expected from the effects of the individual viruses. PVX is, therefore, a potentially damaging pathogen and considerable efforts have been made to breed resistant potato varieties.

Although resistance to infection by PVX has been demonstrated in *S. tuberosum*, other forms of resistance, particularly hypersensitivity (field immunity) and extreme resistance (often misleadingly referred to as 'immunity') have been favoured by plant breeders. This is mainly because resistance to infection is a much more difficult character for the plant breeder to use than the other forms of resistance, which are more simply inherited and are easier to identify in screening tests.

Several potato varieties in Britain (e.g. Epicure, King Edward and Maris Piper) are 'field immune' to PVX because of a hypersensitive response to infection. Infected tissues in such varieties quickly become necrotic, and this stops the spread of PVX within the plant; hypersensitive varieties do not, therefore, become systemically infected. If scions of a systemically infected non-hypersensitive potato variety are grafted on to hypersensitive plants, the latter show a typical 'top necrotic' reaction in which the growing point and possibly

the whole plant is killed. This top necrosis reaction is widely used to test for hypersensitivity to PVX.

Hypersensitivity is race-specific, and Cockerham (1955) distinguished four main groups of PVX strains according to the response which they elicited on varieties carrying the genes *Nx* and *Nb* after grafting tests for top necrosis. Genotypes with *nxnb* (e.g. Arran Banner) are susceptible to strains in all four groups; genotypes *Nxnb* (e.g. Epicure) are resistant to groups 1 and 3 but susceptible to groups 2 and 4; Arran Victory, with the genotype *nxNb*, is resistant to groups 1 and 2 but susceptible to groups 3 and 4; Craigs Defiance, with the genotype *NxNb*, is susceptible only to strains in group 4. The most common strains of PVX occurring in Europe are those of group 3, and many British varieties carry the *Nx* gene which is effective against strains of group 3. Many common European varieties carry the gene *Nb*, but only relatively few (e.g. Pentland Dell and Ambassadeur) carry both the genes *Nx* and *Nb*. Genes controlling hypersensitivity have also been found in wild *Solanum* species, including *S. chacoense* (Yashina, 1974).

Because hypersensitivity to PVX is race-specific, many plant breeders have preferred to exploit 'extreme resistance' which is non-race-specific. Extreme resistance to PVX was first reported in the USA by Schultz and Raleigh (1933) in a potato seedling known as USDA 41956; several varieties including Saco and Taura were derived from this seedling. Other sources of extreme resistance are *Solanum acaule* (a wild species) and the Andigena clone, CPC 1673, also used as a source of nematode resistance. This type of resistance is controlled by a single dominant gene in all these sources, but the resistance gene from *S. acaule* is probably different from the gene in Andigena and USDA 41956. Extreme resistance to PVX has also been found in *S. neo-tuberosum* (Glendinning, 1975) and several other *Solanum* species, including *S. rybinii* (*S. phureja*) and *S. chacoense* (Sokolova and Savchenko, 1974). Resistance has been successfully transferred from many of these sources to early, high-starch breeding material (Dziewońska, 1974). Little is known about the mechanisms of resistance to PVX. In Czechoslovakia, a good association has been reported between the concentration of a virus inhibitor in leaf sap, and resistance to PVX in the field; the concentration of this inhibitor, which may be a protein of low molecular weight, can be increased by certain fertilizer treatments and this could affect the expression of resistance of plants growing under different nutrient conditions (Jermoljev, Bečková and Chod, 1968). In the USA the variety Saco, which has extreme resistance to PVX derived from the USDA seedling 41956, contains a substance which is a powerful inhibitor of this virus (Tsou, Juo and Rich, 1967).

Both hypersensitivity and extreme resistance have been used extensively in breeding programmes, and many varieties which exhibit one of these forms of resistance are being widely grown in Europe and the USA. There are many theoretical disadvantages to the use of hypersensitivity to PVX, particularly its strain-specificity. It should be emphasized, nevertheless, that varieties such as Epicure and King Edward (which carry the hypersensitivity gene *Nx*) have been widely grown for more than 50 years, and their resistance is, apparently, as effective as ever. In spite of this stability of hypersensitive types of resistance in potatoes, breeding for extreme resistance is still to be preferred, because only two genes are necessary to confer simultaneously extreme resistance to PVX, PVY and PVA.

OTHER VIRUSES

Resistance to several viruses other than leaf roll, PVY and PVX has been reported in *Solanum* spp. For example, hypersensitivity to potato virus S is present in an Andigena clone in which it is controlled by one dominant gene (Baerecke, 1967). The variety Saco, which was derived from USDA seedling 41956, expresses good resistance to virus S in addition to extreme resistance to PVX (Bagnall and Young, 1968). Zadina (1971) screened more than 600 potato clones for resistance to potato virus S and found that only Saco resisted all attempts to infect it. Other workers, however, have reported that Saco can be infected, but only with difficulty.

Resistance to tobacco rattle virus also occurs in several potato varieties, but the resistance is strain-specific and its expression in the same variety can vary in different soils. The resistance apparently involves virus tolerance, resistance to virus infection and hypersensitivity.

CONCLUSIONS

Good sources of resistance to most of the important virus diseases of potatoes have been found in *Tuberosum* or *Andigena* varieties or in several wild *Solanum* species. The most important types are resistance to virus infection, hypersensitivity and extreme resistance, although considerable differences in virus tolerance have also been recorded. Extreme resistance ('immunity') is generally the most desirable type of resistance, because it is usually controlled by one or two major genes, and has been effective against all known virus strains over a long period of agricultural use. Hypersensitivity is a very useful type of resistance and is simply inherited, but is probably less desirable because it is strain-specific; this strain-specificity has not been a serious drawback in practice, however, and varieties carrying the hypersensitivity gene N_x have shown good resistance to PVX for over 50 years. Resistance to virus infection can also give a good control of viruses in the field, but it is more difficult to work with in a breeding programme than hypersensitivity or extreme resistance, because testing procedures are complicated and inheritance is polygenic. In the case of leaf roll, however, resistance to virus infection is the only type of resistance that is being widely exploited.

Considerable progress has been made in developing varieties with resistance to one or more of the viruses, leaf roll, PVY, PVX, PVS and tobacco rattle, but there are few varieties with resistance to all these viruses. Other control measures, particularly hygiene, will therefore still be necessary in the future, although it seems that resistant varieties will play an increasingly important part.

Sugar Beet

Although sugar beet can be attacked by many virus diseases, only two, virus yellows and curly top, are of great economic importance. Both of these diseases are caused by insect-transmitted viruses and much effort has been devoted to breeding for resistance to them.

VIRUS YELLOWS

Virus yellows can be caused by one or more of three aphid-transmitted viruses, namely beet yellows virus (BYV), beet mild yellowing virus (BMV) and beet western yellows virus (BWV). All three viruses can be transmitted by *Myzus persicae* but *Aphis fabae*, which also commonly infests sugar beet in many parts of the world, transmits BYV but not BMV or BWV. Although BYV is widely distributed throughout the world it is usually not the most common cause of yellows. For example, BMV is the most prevalent yellowing virus of sugar beet in Europe (Russell, 1958; 1968) and in the USA most virus yellows is caused by BWV (Duffus, 1960). There is considerable evidence that BMV and BWV are distantly interrelated, but neither is related to BYV (Duffus and Russell, 1975). Thus, in breeding for resistance to virus yellows, the plant breeder is dealing with three viruses, each of which exists in a number of strains. The situation is further complicated by the fact that the older leaves of plants infected with BMV are predisposed to attack by secondary fungal pathogens, particularly species of *Alternaria*, which cause premature death of the older leaves (Russell, 1965; 1966c).

In spite of an extensive search within the genus *Beta* for sources of inherited immunity to the three viruses, none has been found. All plants of the genus *Beta* that have been tested have proved to be susceptible to BYV, BMV and BWV when inoculated by large numbers of viruliferous aphids. However, several kinds of resistance to BYV and BMV have been identified. These include (1) resistance to the aphid vectors, (2) resistance to virus infection (tendency to escape infection), (3) virus tolerance, and (4) resistance to virus multiplication. Some of these comprise at least two independently inherited subcomponents, each of which is apparently controlled by several genes (Russell, 1972a, b).

The first attempts to breed for resistance to virus yellows were carried out independently in the UK by Hull (1960) and in the Netherlands by Rietberg (1959) during the late 1940s. They showed that some plants in most of the sugar beet varieties tested were less damaged by virus yellows than were other plants. This work stimulated more intensive work on breeding for resistance to virus yellows in many European countries, including the UK (Russell, 1960; 1972b), the Netherlands (Cleij, 1970), Germany (Koch, 1974), Czechoslovakia (Petrák and Smrží, 1974) and Poland (Filutowicz, 1960) and in the USA (McFarlane and Bennett, 1963). Selection for resistance to virus yellows is now being carried out in all the major sugar-beet growing areas of the world, including Japan.

Most of the breeding work has been concerned with selecting for virus tolerance within sugar beet varieties. Some lines of wild sea beet (*Beta vulgaris* s.sp. *maritima*) have also shown good virus tolerance (Watson and Russell, 1956; Filutowicz and Pawelska-Kozińska, 1973), but these have many undesirable features and are, therefore, difficult to use in breeding programmes. Watson and Russell (1956) and Russell (1960) showed that screening for tolerance to BYV can be carried out effectively in the glasshouse by selecting young infected plants with mild symptoms and large roots. However, symptoms of BMV and BWV are usually indistinct on sugar beet in the glasshouse and selection for tolerance to these viruses is therefore not very effective under glass. For this reason, selection for tolerance has usually been carried out in the field where it is possible to select simultaneously for improved tolerance to BYV, BMV and

BWYV and for resistance to *Alternaria*. Plants that are tolerant to one virus are not necessarily tolerant to the others; it is therefore necessary to select for tolerance to more than one virus, either individually or in combination. The usual practice has been to carry out recurrent selection of individual plants with unusually heavy roots when virus-infected, selected plants generally being either crossed in pairs or interpollinated in groups (Russell, 1964a). The degree of yellowing of the leaves of infected plants is usually a poor guide to the amount of damage caused to the roots by virus infection and is, therefore, of limited use in selecting for tolerance. However, flying aphids are attracted towards orange or yellow objects; virus-infected plants that remain green may therefore be less susceptible to aphid settling, and consequently are less likely to become infected with aphid-transmitted viruses, than are plants which show severe leaf yellowing.

The virus tolerance and general field performance of partially inbred lines that were developed by recurrent selection, were assessed in successive generations and the most promising lines were usually interpollinated to produce 'experimental varieties'. Maris Vanguard, a multigerm virus-tolerant variety, which was grown in areas of eastern England where virus yellows was particularly common during the late 1960s, was produced by interpollinating three partially inbred lines. This variety gave substantially higher yields of sugar than other varieties when infected with virus yellows (see Figure 9.2) and equivalent



Figure 9.2 The effects of virus yellows on Maris Vanguard, a tolerant sugar beet variety (left) and on a sensitive variety (right). (By courtesy of Plant Breeding Institute, Cambridge)

yields when healthy (Russell, 1964a, b; 1972b). However, the roots of healthy and virus-infected plants of Maris Vanguard contained unacceptably low concentrations of sugar and high concentrations of impurities such as sodium and potassium. Maris Vanguard has, therefore, been superseded in Europe by high-quality, monogerm virus-tolerant varieties, most of which have been developed by controlled hybridization between breeding lines using cytoplasmic male sterility. Nevertheless, Maris Vanguard is considered to be a promising source of resistance to virus yellows (Petrák and Římsa, 1977).

In the USA an inbred line, designated 413, was developed from a widely grown multigerm variety, US 75, after five successive generations of selection for virus tolerance in the field, using heavy root weight as a criterion of tolerance. This line has been used as a pollinator for monogerm, male-sterile lines in the production of hybrid, monogerm virus-tolerant varieties, including US H9 and US H10 which have been grown commercially in California (McFarlane and Skoyen, 1968; McFarlane, Skoyen and Lewellen, 1969). In those areas of California where virus yellows is common these varieties have usually given considerably higher yields of sugar than have other varieties, but they are very susceptible to powdery mildew (Kontaxis, 1976) and bacterial rot (Thomson *et al.*, 1973).

Many of the virus-tolerant varieties that have been produced have had unusually low concentrations of sugar and high concentrations of juice impurities in the roots. However, there is apparently no direct or causal relationship between the concentrations of these compounds and virus tolerance, because high-quality virus-tolerant varieties have recently been marketed in Europe. The apparent association between low quality and virus tolerance is probably attributable to the techniques that have usually been employed in selecting for tolerance to virus yellows, particularly the selection of large roots, which tend to have low sugar content.

The genetics of virus tolerance is not properly understood. In some breeding material, tolerance is apparently partly controlled by a dominant gene, although modifying genes are important (Cleij, 1970). However, most workers have found that the progenies of paired crosses between tolerant and sensitive plants show an intermediate level of tolerance, suggesting that tolerance is polygenically inherited. In some reciprocal crosses between certain inbred lines, plants grown from seed harvested from one parent were more tolerant than those grown from seed of the other parent (G.E. Russell, unpublished data); this suggests that cytoplasmic genes may sometimes influence the expression of tolerance to virus yellows. Resistance to *Alternaria* spp., the effects of which are often difficult to distinguish from those of virus tolerance, is inherited as a partially dominant character (Russell, 1965; 1966c), and this has probably contributed to the confusion regarding the inheritance of virus-yellows tolerance.

The possibilities of using types of resistance other than virus tolerance have also been explored. The epidermal cells of sugar beet leaves give a hypersensitive response to sap inoculation of BYV which usually prevents spread of the virus from the inoculation points, although systemic virus spread does occur in a small proportion of inoculated plants. BMV and BWV have not been transmitted by contact inoculation and hypersensitivity to them has not, therefore, been observed. As all three viruses are invariably transmitted by aphids in the field, by means of feeding probes into the phloem, hypersensitivity is of no practical importance in breeding for resistance to virus yellows.

Resistance to virus infection, which is expressed as an inherited tendency to escape infection when exposed to inoculation by viruliferous aphids, has been exploited successfully in breeding programmes (Russell, 1966a, d). Viruliferous aphids feed on plants that express this kind of resistance, but do not readily transmit yellowing viruses to these plants. At the Plant Breeding Institute, Cambridge, plants of partially inbred lines were each infested with three viruliferous aphids in the glasshouse, and those which escaped infection were retained for seed production (*see Figure 9.3*). Selected plants were crossed in pairs and



Figure 9.3 In testing for resistance to sugar beet virus yellows in the glasshouse, viruliferous aphids are transferred by brush from virus-infected plants to healthy sugar beet seedlings. (By courtesy of Plant Breeding Institute, Cambridge)

the progenies subjected to similar tests from which further selections were made. After two or three generations of selection in the glasshouse, inbred lines derived from selected plants were tested in the field under conditions where natural spread of virus yellows was likely to occur. Such field tests showed that considerable improvements in resistance to virus infection had been achieved by these methods. Unfortunately, resistance to BMV was not necessarily associated with resistance to BYV, but several breeding lines expressed resistance to infection with both viruses. A drawback to this type of resistance is that it is most effective when the numbers of viruliferous aphids are small. Resistance to virus infection is, therefore, unlikely to be of great benefit in areas where sugar beet crops are attacked simultaneously by large numbers of viruliferous aphids (McFarlane and Bennett, 1963).

Some sugar beet lines that are resistant to virus infection are also resistant to the aphid vectors (Russell, 1966b; Lowe, 1971). It is unlikely, however, that aphid resistance is of overriding importance in resistance to infection with virus yellows because, although both viruses are transmitted by *Myzus persicae*, some

aphid-resistant lines are resistant to only one of the viruses. Although these two forms of resistance seem often to be closely associated, resistance to virus infection probably involves the establishment of the viruses in the host plant, in addition to the efficiency of the aphid as a virus vector (Russell, 1972a, b; Hills, Shepherd and Wakeman, 1973). Haniotakis and Lange (1974) consider that part of the resistance to virus transmission shown by US H9B is associated with the probing behaviour of aphids on the leaves.

There is some evidence from serological and infectivity tests that BYV reaches much higher concentrations in sensitive than in tolerant plants, particularly in the early stages of infection (Beiss, 1963; Smrč *et al.*, 1974). Leaves of some varieties contain compounds that can inhibit the multiplication of BYV (Trzebinski and Pawelska, 1966; Chod, 1967) but their importance in the resistance of intact plants is uncertain. The biochemical and physiological basis of virus tolerance is, therefore, not understood and is probably very complex. Metabolic disturbances, leading to an increased respiration rate, decreased photosynthesis and reduced movement of solutes in the phloem, are presumably less pronounced in tolerant than in sensitive plants.

There are many strains of beet yellows virus, some of which cause much more severe symptoms than others in beet (Russell, 1963; Björling, 1969). Such 'virulent' or 'severe' strains cause correspondingly severe symptoms on both tolerant and sensitive plants and are not, therefore, true resistance-breaking strains (Russell, 1964b). This apparent non-race-specificity of tolerance has been a very encouraging feature in breeding for resistance to virus yellows. There have been no reports of virus strains that have overcome tolerance to BYV, BMYV or BWYV, and it seems that all kinds of resistance to virus yellows at present being exploited may be non-race-specific.

The value of virus-tolerant sugar beet varieties in reducing yield losses from virus yellows has been clearly demonstrated in many parts of the world, and such varieties can be expected to play an increasingly important part in controlling the disease. However, virus tolerance can give only a partial control of virus yellows damage and more varieties, which also incorporate other kinds of resistance, are therefore being developed. Several varieties and breeding lines which express virus tolerance, resistance to virus infection and resistance to *Myzus persicae* have already been developed, for example Maris Vanguard and a partly inbred line, VT 95, at the Plant Breeding Institute in the UK. On such sugar beet, populations of aphid vectors would be reduced, which would in turn allow resistance to virus infection to operate more effectively in reducing virus spread. If either, or both, of these types of resistance should fail to control the spread of virus yellows for any reason, virus tolerance would minimize losses of yield caused by virus infection.

CURLY TOP

Curly top is the most important disease of sugar beet in the USA to the west of the Rocky Mountains. It is apparently absent from Europe and Asia, although a very similar disease has been reported from Turkey and parts of the Middle East. Curly top is caused by a virus that is transmitted by the beet leafhopper, *Circulifer tenellus*, in which the virus persists for long periods; it does not, however, multiply in the insect vector. In the western USA, beet leafhoppers

overwinter on weeds in hilly, desert regions, migrating in the Spring from their winter hosts to agricultural crops in the adjacent valleys. Curly top virus (CTV) has a very wide host range and many of the overwintering weed hosts can also harbour the virus. In the Spring, therefore, a proportion of the *C. tenellus*, which migrate to the young beet crops, are already viruliferous and these can spread CTV very rapidly within the crops. Infected plants remain stunted, with pronounced inrolling of the margins of the lamina, on which there are numerous warty outgrowths (enations), particularly on the underside of the leaves. Susceptible plants are often killed if they become infected, as young plants, with a virulent isolate of the virus.

The disease was so severe in the western USA in the late 1920s that many growers could not produce worth-while yields and the future of the sugar beet industry in this area was seriously threatened. The industry was saved mainly by the successful breeding of curly top-resistant varieties. Intensive programmes of selecting for resistance to curly top began soon after the end of the First World War, particularly in California and Utah by Carsner (1926) and Esau (1930). In early attempts to breed for resistance, individual plants were selected in commercial beet crops, because they appeared to be less severely injured by curly top than other plants. Selected plants were then interpollinated to provide seed for further tests. Two of the many difficulties encountered were that it was not possible to predict where severe attacks of curly top would occur, and that the disease often was not uniformly spread within a crop. Nevertheless, selection for resistance was successfully accomplished, and plants of several promising lines were interpollinated to produce seed of the first curly top-resistant variety, US No.1, which was much more resistant to curly top than the parent varieties. In a field experiment where curly top was severe, US No.1 yielded over 14 tonnes of roots per hectare, whereas the parent variety gave a negligible yield.

Murphy (1942) showed that selecting and testing for resistance to curly top was much more effective when plots of breeding lines were sown between plots of very susceptible varieties in fields adjacent to the leafhopper breeding grounds. Plants that had been infected with curly top in the previous year were transplanted into plots of susceptible material to provide foci of infection. The migratory leafhoppers transmitted curly top virus first to the plants of the susceptible variety, and then to those in adjacent plots of breeding material. The leafhoppers generally multiplied so quickly on the curly top-susceptible varieties that the experimental plots were subjected to a uniform, severe exposure to virus infection and every experimental plant became infected. Plants that suffered least damage in each plot were selected and used for seed production, and the resistance of the progenies was evaluated in subsequent years. In this way, breeding lines with improved resistance to curly top were developed and several new curly top-resistant, open-pollinated varieties were produced from these. The best of these varieties, which resulted from mass selection for resistance, included US 22/4, which gave a much higher yield of sugar than that of earlier curly top-resistant varieties, when infected with CTV.

Attempts have also been made to screen for resistance to CTV in glasshouse tests using young plants, and techniques have been developed in the USA whereby reproducible assessments of resistance can be made in the glasshouse. In particular, a method for mechanically inoculating large numbers of seedlings has recently been devised (Mumford, 1972). Thus, in future it may be possible

to carry out in the glasshouse much of the testing and selection work that, up to now, has been carried out in the field.

In some areas of the USA it has been necessary to breed for resistance to both curly top and leaf spot disease (*Cercospora beticola*). Breeding lines with resistance to both diseases have been produced by crossing *Cercospora*-resistant lines with curly top-resistant lines and backcrossing to the virus-resistant lines (Gaskill *et al.*, 1967). Progenies were then tested for resistance to both diseases and re-selections were made from them. In parts of California, where virus yellows can cause serious losses of yield, it was very desirable to develop curly top-resistant varieties that were resistant to this disease also. Successive selections made from a curly top-resistant variety, US 75, for improved resistance to virus yellows, resulted in the development of an inbred line designated '413' which suffers only about 50 per cent as much damage from virus yellows as the parent variety. This line was used as a pollinator for male-sterile monogerm hybrids derived from lines with good resistance to curly top (McFarlane, 1969). Monogerm varieties with curly top resistance have also been produced by many sugar beet companies in the western United States.

Numerous strains of CTV have been reported to occur in sugar beet (Giddings, 1959), and some of these are so virulent on sugar beet that they cause severe damage on even the most resistant varieties (Bennett, 1963). Virulent strains of CTV have become more common in recent years (Magyarosy and Duffus, 1977), and there is an obvious need to breed varieties with an even higher level of resistance than that of varieties in current use. There is no suggestion, however, that these are 'resistance-breaking' virus strains. Strains of CTV have been very stable in sugar beet and there is no evidence that resistant curly top sugar beet varieties have influenced the distribution or prevalence of individual virus strains (Giddings, 1959). Resistance to curly top in sugar beet has, therefore, been durable and may be non-race-specific.

Although the practical results of breeding for resistance to curly top in sugar beet have been so successful, there is very little published information about the nature or inheritance of resistance. The results of field experiments suggest that virus tolerance is probably an important component of resistance. This is to be expected, because most of the widely used selection methods preclude selection for the expression of disease escape mechanisms. Under less extreme conditions, however, in the presence of small numbers of viruliferous vectors, resistance to the leafhopper vectors and resistance to virus infection might help to decrease or delay the spread of CTV within sugar beet crops.

Although several studies of the inheritance of resistance have been carried out (e.g. Abegg and Owen, 1936; Savitsky and Murphy, 1954) no satisfactory explanation of the genetics of resistance to curly top has been provided (McFarlane, 1969). Resistance appears to be a dominant character when resistant plants are subjected to mild curly top exposure, but not where plants are tested under more extreme conditions of infection. These results suggest that several independently inherited types of resistance to curly top may be present in sugar beet, some of which are expressed only when few viruliferous vectors are present. No differences in susceptibility to CTV were observed between 13 inbred lines of sugar beet with cytoplasmically controlled male sterility and their male-fertile equivalents (Theurer and Mumford, 1976); there is, therefore, no evidence that cytoplasmic factors are important in determining resistance to curly top.

CONCLUSIONS

In sugar beet there are two good examples of successful breeding programmes for the control, by resistant varieties, of two very damaging insect-transmitted virus diseases. If curly top-resistant varieties had not been developed it is very doubtful whether the sugar beet industry could have flourished, or even survived, in the western States of the USA. Progress with breeding for resistance to virus yellows has been less spectacular than with curly top, but resistant varieties have been developed and their use will undoubtedly play an important part in reducing losses from this disease. These breeding programmes have been successful in spite of the fact that resistance to both diseases is very complex and very little is known about the genetics of resistance or the mechanisms that are involved. This shows that considerable agricultural benefits can result from a determined but empirical approach to breeding for resistance. The resistance, perhaps because of its very complexity, seems to be largely non-race-specific and it is, therefore, unlikely that sugar beet breeders will be faced with a major 'breakdown' in resistance caused by new strains of CTV or of the viruses that cause virus yellows. A much higher level of resistance to curly top is needed if major damage from the disease is to be avoided when virulent strains of CTV are present. Until varieties with greater resistance can be developed, it will be necessary to reduce the vector populations, in and around threatened crops, with insecticides.

Rice

Virus diseases of rice cause considerable losses of yield in most of the major rice-producing areas of world. For example, hoja blanca (white leaf) caused by the hoja blanca virus is very damaging in parts of Central and South America, the tungro virus is responsible for serious losses in South-east Asia, and stripe disease can be severe in Japan. Although it is now thought that stripe may be caused, not by a virus but by a mycoplasma, this has not been conclusively proved; resistance to this disease will be considered, therefore, in this Section, together with resistance to the hoja blanca and tungro viruses. Resistant varieties have played an important part in the control of virus diseases of rice (Toriyama, 1969).

HOJA BLANCA (WHITE LEAF)

The hoja blanca virus has characteristic filamentous particles and is transmitted by several species of Hemiptera, including *Sogatodes oryzicola* and *S. cubanus* (Atkins and Adair, 1957). Beachell and Atkins (1959) tested several thousands of rice varieties and lines for resistance to hoja blanca in the field in Cuba and Venezuela during the late 1950s; all the American varieties that were tested were highly susceptible, but *japonica* types of rice were resistant. A breeding programme was then started in the USA by Beachell and Atkins (1959) using several different sources of resistance, including two *japonica* lines from Taiwan (PI 215936 and CI 9416) and two varieties, Lacrosse and Gulfrose. Some of the most promising hybrid varieties which were developed in this programme were

later tested in Cuba, Venezuela, Colombia and Costa Rica, where several varieties showed good resistance to hoja blanca and good agronomic characteristics. Beachell and Jennings (1961) showed that resistance to hoja blanca is simply inherited and dominant.

Trujillo (1969) developed a glasshouse technique for evaluating resistance to hoja blanca infection, the results of which correspond closely to those from field experiments. One viruliferous vector was caged on each test plant at the two to three-leaf stage and was allowed to feed for six days. The insects were then killed by insecticides and the proportion of infected plants in each variety or line was recorded. Very different levels of infection were found in the 16 varieties tested; over 90 per cent of plants in the most susceptible varieties became infected whereas, in the most resistant varieties, which included Lacrosse and Gulfrose, only between 10 and 15 per cent of plants became infected. These results suggest that resistance to virus infection is the main type of resistance involved in varieties such as Lacrosse and Gulfrose, although some lines may be resistant to the insect vectors also. However, these tests were effective in detecting differences in resistance to hoja blanca only when small numbers of vectors were used to inoculate each test plant. This situation is similar to that found in sugar beet, where resistance to infection with aphid-transmitted yellowing viruses is effective only when small numbers of vectors are present (see page 241–242).

Very good progress in breeding for resistance to hoja blanca has been made in Colombia (Ou and Jennings, 1969). Commercial varieties which show good resistance to hoja blanca have been released, and it has not been difficult to combine resistance with other desirable agricultural characteristics. Many varieties and breeding lines from the International Rice Research Institute (IRRI) have recently been tested in Cuba for resistance to hoja blanca virus, and several have been found to be highly resistant (Pérez Ponce *et al.*, 1974).

TUNGRO DISEASE

Tungro is probably the most important virus disease of rice in South-east Asia where it is widely distributed, having been reported from the Philippines, Malaysia, Indonesia, Pakistan, Thailand and India. In 1971 and 1972 it was responsible for particularly severe damage in the Philippines. It is caused by rice tungro virus, which is transmitted in the semi-persistent manner by several species of leafhoppers including *Nephotettix virescens* (*N. impicticeps*), *N. nigropictus* (*N. apicalis*) and *Rocilia dorsalis* (Rivera and Ou, 1965); it is not sap-transmissible. The type of symptom varies with variety; for example, in the variety Taichung (Native), infected seedlings show stunting, mottling and chlorosis of the leaves, whereas seedlings of the variety FK135 show yellow striping with the severe 'S' virus strain and mottling with the milder 'M' strain.

Screening test procedures have been described by Ling (1969). Seedlings are grown in pots and infested at the two- to three-leaf stage with two or three viruliferous insects; plants are examined for tungro symptoms after 12 days and the percentage of successful transmissions in test plants of each variety is recorded. The height of infected plants is compared with that of healthy plants of the same variety, to assess the response of varieties to infection. Many resistant varieties have been found, including several that are currently grown in South-east Asia, and these have been used as sources of resistance by breeders in the

Philippines (International Rice Research Institute, 1973) and in India (Shastri, John and Seshu, 1972). An Indian variety, Pankhari 203, has shown particularly good resistance to tungro (Reddy, 1973) and another, Habigonj, is reported to be almost equally resistant. Field tests have been carried out in areas where the presence of virus sources and of the leafhopper vectors can be expected to lead to severe natural epidemics of tungro. Epidemics can be induced, or encouraged to develop more quickly, by interplanting rows of inoculated plants of susceptible varieties between rows of the potential varieties or breeding lines to be tested (Anjaneyulu, 1975).

Resistance to *Nephotettix virescens*, the principal vector of tungro virus, has been studied by Pathak, Cheng and Fortuno (1969); this work will be discussed in detail elsewhere in this book (see page 344). Pankhari 203 is highly resistant to *N. virescens*, larvae of this insect growing very slowly and having an unusually low survival rate on plants of this variety. A dwarf variety, IR 8, which has become widely grown because of its high yield, is also resistant to this leafhopper. Resistance to *N. virescens* is a dominant character, being controlled by a series of single dominant genes (International Rice Research Institute, 1973).

In mass screening tests at IRRI, varieties have been classified into three main groups according to their susceptibility to infection with tungro virus: (1) resistant (less than 30 per cent infection); (2) intermediate (30–60 per cent infection); and (3) susceptible (more than 60 per cent infection). Varieties which have been regarded as resistant in these tests have subsequently shown a high level of resistance to tungro virus in the field. Virus tolerance has also been assessed at IRRI by estimating the reduction in plant height caused by infection. Sensitive plants can show a reduction in height of more than 75 per cent, but infected tolerant plants may show no reduction in height.

Although preliminary experiments suggested that resistance to tungro virus is inherited as a dominant character, more recent work has shown that the genetics of resistance is complex. Seetharaman, Prasad and Anjaneyulu (1976) have reported that resistance in three varieties is controlled by three dominant genes and an inhibitory gene. This complexity is to be expected because resistance to tungro probably involves resistance to the vectors, resistance to virus infection and tolerance, each of which may be inherited independently and probably involves quite distinct resistance mechanisms. Very little is known about the nature of resistance, but it has been reported that tungro infection decreases the concentrations of chlorophyll, protein and RNA less in virus-tolerant varieties, such as IR 8, than in sensitive varieties such as Padma (Chowdhury and Mukhopadhyay, 1974).

Several rice varieties that are highly resistant to tungro in the Philippines express less resistance in India, suggesting that resistance is race-specific. Although at least three strains of tungro virus have been identified (Rivera and Ou, 1967; Mishra *et al.*, 1976) such apparent breakdowns in resistance may be attributable to higher populations of insect vectors in some areas than in others, rather than to resistance-breaking virus strains.

STRIPE

Stripe is a serious disease of rice in parts of Japan and South Korea. There is some doubt about whether the causal pathogen is a virus or a mycoplasma-like organism (MLO); for convenience it has been included in this section. The stripe

pathogen is transmitted by several species of leafhopper, including *Leodelphax* (*Delphacodes*) *striatellus* and *Unkanodes sapporonus*. Infected plants show chlorotic striping or general chlorosis of the leaves and they are severely stunted; leaves die prematurely and infected plants characteristically droop. Even if diseased plants survive they usually produce little grain particularly if they became infected at an early growth stage. In resistant varieties, symptoms are much less severe and consist of slight chlorosis and mottling of the leaves.

Work on breeding for resistance to stripe has been confined mainly to Japan, although screening of varieties and breeding material for resistance has now been started in Korea (Chung, 1974). Varietal differences in resistance to stripe were first observed in field experiments in Japan by Suzuki *et al.* (1960). Although most screening for resistance has been carried out in the field, Sakurai and Ezuka (1964) developed a method for screening large numbers of seedlings in the laboratory. In these tests, seedlings were grown in Petri dishes filled with soil and were infested with larvae of *L. striatellus* that had been reared on stripe-infected plants. Seedlings that were very sensitive to stripe died, or became severely stunted, whereas resistant seedlings grew fairly strongly.

Sakurai and Ezuka screened more than 400 Japanese and introduced varieties using this seedling test method, and found that *indica* and Japanese upland varieties were generally much more resistant to stripe than the lowland varieties. Later experiments showed that most varieties from Burma, China, India, Indonesia, Pakistan, the Philippines, the USSR and Vietnam were also resistant. This may explain why stripe is a serious disease only in Japan, and in some neighbouring countries where susceptible varieties are very widely cultivated. A hybrid selection, designated St1, which was derived from a cross between Modan (a resistant *indica* variety from Pakistan) and Norin 8 showed an unusually high level of resistance. St1 was repeatedly backcrossed with Norin 8 to give Chugoku 31, a variety resistant to both stripe and blast. Although Chugoku 31 is a low-yielding variety it has been widely used in subsequent breeding programmes (Toriyama *et al.*, 1966). For example, Mineyutaka (= Norin 225), which has been developed from a cross between St1 and Sachikaza, has good resistance to stripe and several other diseases, including blast and bacterial blight (Toriyama *et al.*, 1972).

Resistance to stripe has been reported by Toriyama (1972) to be controlled by an allelic series of genes at the *St*₂ locus which confer different levels of resistance, and also by gene *St*₁ which enhances the expression of resistance controlled by the *St*₂ genes. Yamaguchi, Yasuo and Ishii, (1965) showed that the expression of resistance can be influenced by the female parent, indicating that cytoplasmic genes are also involved in the inheritance of stripe resistance.

As with tungro, there are probably at least three types of resistance to stripe, including non-preference of the vectors for certain plants, resistance to the development of disease, and tolerance (Yamaguchi *et al.*, 1965). Okamoto and Inoue (1967) found that some stripe-resistant rice varieties are resistant to the insect vectors of stripe, and Toriyama (1972) found that the leafhopper *L. striatellus* prefers to feed on and colonize plants of the stripe-susceptible *japonica* types rather than on the more stripe-resistant *indica* varieties. These and other results suggest that vector resistance may be an important component of resistance to stripe.

OTHER VIRUSES

Several other virus diseases of rice are of considerable local importance. Two examples of such diseases are rice dwarf and black-streaked dwarf, both of which have occasionally caused serious losses of yield in Japan. Dwarf is caused by a virus which is transmitted by the leafhoppers *Nephotettix virescens* and *N. nigropictus*; it is passed in these insects from generation to generation through the eggs. Yasuo, Yamaguchi and Ishii (1960) found that most Japanese varieties are very susceptible to dwarf but that many foreign varieties are resistant.

Black-streaked dwarf is also transmitted by leafhoppers, including *Leodelphax striatellus*. Morinaka and Sakurai (1966) developed a method of screening large numbers of seedlings for resistance to the disease. They tested many varieties and reported that several show some resistance.

CONCLUSIONS

Although breeding for resistance to virus diseases in rice is a comparatively recent venture, most of the important work having been carried out since 1960, considerable progress has been made. Sources of resistance to all the major virus diseases have been found, and these are being exploited by plant breeders in many parts of the world. Resistance to these diseases involves resistance to the insect vectors and resistance to virus infection, as well as virus tolerance. In spite of the apparent complexity of resistance to hoja blanca, tungro and stripe, resistance to each disease seems to be dominant and controlled by a few major genes. Although there are probably many different strains of each of the pathogens, resistance-breaking strains do not seem to have created major problems in the field.

Tobacco

Tobacco can be attacked by a number of different viruses, the most important of which are probably tobacco mosaic virus (TMV), cucumber mosaic virus (CMV), potato virus Y (PVY), tomato spotted wilt virus (TSWV) and tobacco etch virus (TEV) (Lucas, 1965; Akehurst, 1968). Disease resistance has been a very important factor in the breeding of new tobacco varieties. Considerable effort has been devoted to breeding for such resistance, with some success in developing varieties that are resistant to TMV, CMV, PVY, TSWV and TEV.

TOBACCO MOSAIC

Tobacco mosaic virus (TMV) is the most widespread, and probably the most damaging, of all the viruses that can infect tobacco. Its importance can be judged by the fact that in 1966 tobacco mosaic caused an estimated \$2.8 million worth of damage to the flue-cured crop in North Carolina alone.

The most usual symptoms caused by TMV are mottling of dark and light green patches on the leaves, although the intensity of these mosaic symptoms

and the extent of chlorosis and necrosis of infected leaves, depend very much on the strain of virus and the host genotype involved. The disease can cause severe stunting of the plant, particularly if the infection occurs in young plants but, even where stunting is not severe the curing quality of the leaves can be adversely affected. No specific vector of TMV is known and the virus is spread almost entirely by contact within a crop, either by direct contact between plants, or by workers or implements during normal cultivation procedures. In susceptible plants the virus quickly becomes systemic. Three main types of resistance to TMV, tolerance, hypersensitivity, and resistance to infection, have been reported.

In the early 1930s a Colombian tobacco variety, Ambalema, was found to be resistant to TMV (Nolla and Roque, 1933); plants of this variety can be infected with TMV but the mosaic symptoms on the leaves are so mild that the plants are almost unharmed. This virus tolerance is controlled by a pair of independently segregating recessive genes which have been designated *rm1* and *rm2*. Holmes (1955) showed that Ambalema is less easy to infect with TMV than most other tobaccos, and that infected leaves of this variety contain low concentrations of virus; movement of TMV into young developing leaves is also delayed in Ambalema. Bancroft and Pound (1954) also found that Ambalema shows hardly any symptoms when infected with TMV, and contains low virus concentrations when infected plants are grown at temperatures between 16 and 20°C. However, plants of Ambalema develop severe TMV symptoms and contain high concentrations of virus when grown at 28°C. The Ambalema type of resistance is therefore temperature-dependent and involves resistance to virus multiplication and spread, in addition to virus tolerance.

Ambalema is not a high-yielding or high-quality variety, and many attempts have been made to introduce the Ambalema type of resistance into more desirable genetic backgrounds. No commercially acceptable varieties have yet been developed from such breeding programmes, and it seems that the exploitation of TMV resistance from Ambalema is hampered by undesirable linkage and pleiotrophic effects. For example, TMV-resistant Ambalema types are particularly prone to wilting. For these reasons, breeding for the Ambalema type of resistance to TMV has been largely abandoned in favour of other forms of resistance (e.g. Hartana, 1969).

Nicotiana glutinosa is hypersensitive to TMV, and necrotic local lesions develop on the leaves after mechanical inoculation with this virus; under most conditions the virus is restricted to these lesions but when inoculated plants are grown at temperatures of over 30°C, TMV becomes systemic. At normal field temperatures *N. glutinosa* is 'field immune' to TMV; this type of resistance has been successfully used in many tobacco breeding projects throughout the world. Holmes (1938) showed that hypersensitivity to TMV is controlled by a single dominant gene (designated *N*) in *N. glutinosa* and the gene has been transferred to *N. tabacum* in which it confers hypersensitivity (Henderson, Sears and Spassof, 1957; Apple, Chaplin and Mann, 1962). The resistance mechanisms are not understood, but phosphorus-containing antiviral compounds have been identified near TMV-induced lesions in resistant plants (Antignus, Sela and Harpaz, 1975). A fertile amphidiploid species, *N. digluta*, has been produced from crosses between *N. glutinosa* and *N. tabacum*, and repeated backcrosses to *N. tabacum* have enabled the *N* gene to be incorporated into the genetic background of many tobacco varieties.

Fewer TMV lesions are formed on inoculated plants of some tobacco varieties

and breeding lines than on those of others. For example, the line TI 245 shows a marked resistance to infection with TMV and several other viruses. This resistance is controlled by two genes (Holmes, 1960) and involves a tendency to escape infection and a restriction of virus spread. The resistance of TI 245 is associated with an abnormally low number of ectodesmata on the leaf surface (Thomas and Fulton, 1968a, b), which suggests that there may be relatively few entry points for viruses on the surface of leaves of this line. Antiviral factors may be responsible for the restriction of virus movement in the leaves of TI 245. A similar tendency to escape TMV infection has been observed in certain other tobaccos.

Holmes (1955) produced experimental tobacco lines which are highly resistant to TMV and which express hypersensitivity, resistance to virus infection, and tolerance. Although considerable yield increases have resulted from the cultivation of TMV-resistant varieties, such varieties have usually been less productive when grown under virus-free conditions (Chaplin, 1970). However, plant breeders should eventually be able to develop TMV-resistant varieties of good quality and with satisfactory yields. There is no evidence that resistance-breaking strains of TMV have overcome any of the types of resistance that are being used in breeding programmes.

CUCUMBER MOSAIC

More than 300 tobacco varieties and *Nicotiana* species were screened for resistance to cucumber mosaic virus (CMV) in Russia during the early 1960s (Daškeeva, 1965) but no immunity or extreme resistance was found. Hypersensitivity to CMV has been identified in *Nicotiana benthamiana*, *N. bonariensis* and *N. raimondii*, and some hybrids between these species have expressed resistance to virus infection. Ternovskij and Podkin (1970) reported that there is an unusually long incubation period between inoculation with CMV and the first appearance of symptoms in certain species of *Nicotiana*, including *N. tomentosa*, *N. tomentosiformis*, *N. otophora* and *N. raimondii*; symptoms were generally milder in these species than on most *Nicotiana* spp. Crosses between amphidiploids derived from these species and *N. tabacum* showed that this resistance is a recessive character.

A programme of breeding for resistance to CMV is also in progress in Taiwan (Wan *et al.*, 1971). Seven resistant plants were selected from more than 39 000 F₂ plants derived from crosses between two varieties, GAT₂ and GAT₄, with Hicks Broadley, a high-yielding, good quality variety. Re-selections have been made from progenies of recurrent backcrosses with Hicks Broadley in attempts to obtain resistant varieties with high yields and good quality.

The resistance to virus infection of the tobacco line TI 245 has already been mentioned in relation to TMV (*see above*). Troutman and Fulton (1958) showed that TI 245 tends also to escape infection with CMV and that the local lesions which do develop are much smaller than those on more susceptible varieties. This resistance is apparently controlled by two genes and seems to be non-race-specific because it is effective against at least six viruses. This type of resistance does not seem to have been fully exploited by breeders.

Most control measures have involved the control of the aphid vectors of CMV, either directly by insecticides or indirectly by means of barrier crops.

In the future, however, there seem to be excellent prospects of controlling CMV by resistant varieties.

VEIN BANDING

Vein banding, a disease caused by the aphid-transmitted potato virus Y (PVY) is common in many of the tobacco-growing areas of the world, but is particularly serious in Europe. One of the reasons for its importance in Europe is that most of the varieties which are grown there are resistant to blue mould (*Peronospora parasitica*) but are very susceptible to PVY. Resistance to PVY is monofactorially dominant (Van Der Ven, 1966) and, although resistance to each of the two diseases is inherited independently, the two factors are probably closely linked. Partial resistance to PVY infection has been observed in a mutant derived from the variety Virginia A, this resistance being controlled by a single, recessive gene (Koelle, 1962). Little is known about the resistance mechanisms involved. Varieties which are resistant to PVY have a higher polyphenol oxidase activity than susceptible varieties but this relationship is probably indirect. The types of resistance that have so far been identified seem to be very race-specific, and this has limited their usefulness.

As with cucumber mosaic virus, resistant tobacco varieties have not played an important part in the control of vein banding and they are unlikely to do so in the near future. The main control measures will, therefore, continue to be to reduce the number of sources of infection and to control aphid populations by pesticides.

TOMATO SPOTTED WILT

Tomato spotted wilt virus (TSWV) which is transmitted by species of thrips, particularly *Thrips tabaci*, is an important disease in some major tobacco-growing areas, but not in the USA or Rhodesia. Berbeć and Opoka (1966) reported that *Nicotiana glauca* is resistant to TSWV and that hybrids between this species and *N. tabacum* are also resistant; this resistance is inherited as a dominant factor. A resistant line was selected from the F₂ plants of a *N. tabacum* × *N. glauca* cross for further breeding work.

There are also indications of tolerance to TSWV in some tobacco varieties. According to Akehurst (1968), some plants show an ability to grow out of the disease, and exhibit progressively less severe symptoms as the plant grows older. However, although there are good prospects for breeding resistant tobacco varieties, resistance to TSWV has not been given a high priority in breeding programmes.

TOBACCO ETCH

Tobacco etch virus (TEV) has been reported from the USSR, the USA and Canada, where its effects have been particularly damaging in Ontario in some years. Different varieties respond to TEV with different types of symptoms; for example, severe necrosis of the leaves is common in Burley varieties, whereas

mild chlorotic mottling and vein clearing are the main symptoms in most other varieties. These other varieties have a dominant gene which is lacking in Burley tobaccos, and this seems to confer some resistance to TEV. Gooding (1970) found that an American tobacco line, NC 2512, showed good tolerance to TEV and lost only about 3 per cent of its potential yield through infection; this was considerably less than the losses in other varieties examined. The severity of symptoms shown by the varieties did not always reflect the amount of yield reduction caused by the disease. Haslam (1965) has also reported tolerance to TEV in a variety, Harwin, in Canada.

Cocoa

Swollen shoot is the only serious virus disease of cocoa.

SWOLLEN SHOOT

Swollen shoot was first observed in Ghana in 1936 and its causal virus was later identified by Posnette (1947). It is widespread in West Africa and has also been reported from Ceylon. The virus is transmitted by many species of mealy bugs of which *Planococcoides njalensis* (Laing) is probably the most important; the virus is stylet-borne and semi-persistent in the vector. There are many different strains of cocoa swollen shoot virus (CSSV) and the symptoms of swollen shoot vary greatly according to the virus strain involved. Most strains cause chlorosis and some, necrosis, of the leaves and significantly decrease yield, but trees are rarely directly killed by virus infection. Other strains, such as that designated 'New Juaben', cause severe swelling of the stems and tap roots in addition to vein-banding and chlorosis of the leaves; plants infected with such strains rarely survive for more than three or four years. The disease causes extensive damage in Ghana and Nigeria, a total of more than 60 million diseased trees having been destroyed between 1946 and 1957 in Ghana alone. In the 1940s Posnette and Todd (1951) compared the resistance to swollen shoot of Amelonado, the most popular West African variety at that time, with that of several introduced South American varieties. Seedlings of many varieties from the Upper Amazon region of Ecuador, particularly those from the Nanay peninsula, were much more resistant to swollen shoot than were those of Amelonado. A large part of this resistance involves resistance to virus infection, although considerable differences in virus tolerance were also observed. Accordingly, many Amazon varieties were introduced into West Africa for breeding purposes (Knight and Rogers, 1955).

Longworth and Thresh (1963), and Legg and Lockwood (1977), have confirmed in Nigeria that Amelonado clones are very susceptible to swollen shoot virus and that some Upper Amazon cocoas, including Nanay, express good resistance. Some Scavina clones, however, are very susceptible and many Parinari clones show a veinal necrosis that causes infected leaves to collapse and die. Progenies of crosses between swollen shoot-sensitive and tolerant clones are themselves sensitive, showing that tolerance is a recessive character.

In Ghana, Attafuah and Glendinning (1965a) studied the resistance to swollen shoot of the progeny of a tree designated T17, which had been exceptionally difficult to infect with CSSV. Tree T17 had been derived in Trinidad from an

open-pollinated pod on a tree of Iquitos, which had descended from another South American variety, Amazonia Forastero. Cuttings of T17 and of Amelonado were each infested with about 50 viruliferous mealy bugs, which were then allowed to feed on the plants for three days. The proportions of infected plants in each variety were recorded and infected plants were scored for vigour. There was some evidence of resistance to virus infection in the progeny of T17, which was also considerably more tolerant than Amelonado. In a later paper, Attafuah and Glendinning (1965b) described tests in which cuttings of 27 cocoa selections were infested with viruliferous mealy bugs. Differences in resistance to virus infection and tolerance were observed between these selections, clones derived from Iquitos being particularly tolerant, and some other selections were found to express resistance to infection.

In Ghana an intensive programme of disease eradication was put into operation in the 1940s, growers being encouraged to burn all diseased trees. As it would probably be more difficult to identify diseased plants of tolerant varieties, virus tolerance has not been considered a desirable objective in breeding programmes by many breeders in that country. In Nigeria, however, where a similar eradication programme was abandoned in the early 1960s, tolerance to swollen shoot virus is considered to be a potentially useful attribute of varieties.

Kenten and Legg (1970) developed a method of screening for resistance to CSSV using contact transmission instead of grafting or insect transmission. Beans were manually inoculated with a purified preparation of a virulent strain of the virus, and the proportions of successful inoculations in different cocoa types were subsequently recorded. The results were compared with those obtained by mealy bug transmission. Both methods of inoculation resulted in more infection and more severe symptoms in Amelonado cocoas than in the Nanay Amazon types, thus confirming the results of earlier workers. Hybrids between Amelonado and Nanay types showed an intermediate level of resistance to virus infection and tolerance; this indicated that both types of resistance are quantitatively inherited. Kenten and Legg (1971) concluded that the manual inoculation method of screening for resistance to virus infection gives more reproducible results, and is easier to carry out, than conventional tests involving inoculation by mealy bugs.

Using this manual inoculation method, Legg and Kenten (1971) tested a number of cocoa progenies for resistance to virus infection and tolerance, and confirmed the resistance of several Upper Amazon types, including Nanay and Iquitos. Progenies derived from crosses involving the Scavina, Iquitos and Nanay cocoas showed the greatest resistance to virus infection. For example, 75 per cent of parent Amelonado trees were infected with swollen shoot after 41 months compared with only 22 per cent in derivatives of resistant Scavina clones. The Amazon varieties were more tolerant than Amelonado when tolerance was assessed by measuring the reduction in dry weight of cocoa per pod, caused by virus infection.

Differences between cocoa progenies in resistance to mealy bugs have recently been reported (Bigger, 1975). Such differences could not have been detected in screening tests using contact transmission. Methods of testing young cocoa plants for resistance to CSSV using viruliferous mealy bugs have, therefore, been developed involving insect-proofed 'gauzehouses', constructed of timber and muslin-like material (Legg and Lockwood, 1977). The results of such tests have been positively correlated with those from field tests, which can take six to eight years to carry out. Resistance to several strains of CSSV has

been demonstrated in cocoa populations using gauzehouse tests (Kenten and Lockwood, 1977). The resistance has been stable under a range of different inoculation pressures. The best sources of resistance have been Nanay and Iquitos populations and certain Trinitarios types. The level of resistance can be built up by accumulating resistance factors from these different sources (Kenten and Lockwood, 1977).

CONCLUSIONS

Cocoa swollen shoot is primarily a West African disease, perhaps because the Amelonado types of cocoa, which have been widely used there, are unusually susceptible to the disease. Many of the varieties that are used in other areas show a moderate level of resistance, and it seems unlikely that cocoa swollen shoot will cause great damage outside Africa. The introduction into West Africa of resistant breeding material from the Upper Amazon area of South America may eventually lead to effective control of swollen shoot by resistant varieties (Kenten and Lockwood, 1977). Several Amazon varieties show good resistance to virus infection and virus tolerance; selected progenies of these introductions and of crosses between them will be of great value in areas of Ghana and Nigeria where swollen shoot is particularly damaging. A combination of resistance to virus infection and virus tolerance would probably greatly reduce both the incidence of swollen shoot and the damage caused by the disease. However, virus tolerance might make it more difficult to eradicate swollen shoot by destruction of infected plants. An alternative approach might be to combine resistance to CSSV infection with sensitivity (intolerance) to this virus. Such a combination, which is apparently already present in some Scavina cocoas, might restrict and delay the spread of swollen shoot and, at the same time, permit a disease eradication scheme to be carried out.

Tomato

The tomato crop is susceptible to many virus diseases, of which tomato mosaic, curly top and spotted wilt are of particular importance. Resistant varieties have played a major part in the control of these diseases (Walter, 1967).

TOMATO MOSAIC

Tomato mosaic, which is caused by tobacco mosaic virus (TMV), can cause a decrease in plant vigour, loss of yield and poor fruit quality in glasshouse crops of tomato. Although various methods of control, especially improved hygiene, have been used with some success, the growing of TMV-resistant varieties will probably be the most promising control measure in the long term. Plants which resist TMV infection can show yield increases of up to 37 per cent (Pelham, Fletcher and Hawkins, 1970). Programmes of breeding for resistance were first started in the USA in the late 1930s, but it was not until more than twenty years later that similar work was begun in Europe. Early work on breeding for resistance to TMV in tomato has been fully reviewed by Pelham (1966) and only a brief outline of these early investigations will, therefore, be given here.

Three main types of resistance to TMV in tomatoes, namely tolerance, resistance to virus infection and hypersensitivity, have been used in the development of resistant varieties. These types of resistance often occur together in a single plant and it is not always possible to distinguish between them; the different types of resistance may even be controlled by the same, or closely linked, genes.

Virus tolerance was first reported to occur in *Lycopersicon hirsutum* lines from South America in the 1930s. A programme of hybridizing these lines with *L. esculentum* lines and varieties was quickly started in the USA and lines with good tolerance to TMV, apparently controlled by three recessive genes, were developed (Holmes, 1939). Tolerance to TMV has also been found in other species of *Lycopersicon*, including *L. peruvianum*, *L. pimpinellifolium* and *L. chilense* (Frazier *et al.*, 1946). In hybrids between these species TMV symptoms are often delayed and mild; this suggests that they express both resistance to virus multiplication and tolerance. The resistance of these species was at first reported to be oligogenically controlled, but later work suggested that the inheritance of resistance is more complex. Resistance to virus infection was also found in *L. chilense* by Holmes (1939) and this resistance has been introduced into *L. esculentum* lines.

Allelic genes at two loci seem to control most of the resistance to TMV that is expressed by these wild *Lycopersicon* species. One gene, designated *TM-1*, controls resistance to TMV infection and virus tolerance; another gene, *TM-2* and an allele, *TM-2²*, which are located on chromosome 9 (Pelham, 1969), control hypersensitivity. *TM-2* is linked with an undesirable recessive gene which causes severe stunting and chlorosis in the homozygous condition. Cirulli and Alexander (1966; 1969) showed that, although the expression of resistance controlled by *TM-2²* is completely dominant with five strains of TMV at 20°C, F₁ hybrids between resistant and susceptible plants show mild necrosis with three of these virus strains, and severe necrosis with another, when they are grown at 30°C; the hybrids are resistant to the fifth TMV strain at both temperatures. These results, and those of Cirulli and Ciccamese (1975) show that the expression of resistance to TMV is very much dependent on a genotype—environment interaction. This helps to explain the many conflicting reports from different workers concerning the genetics of resistance to TMV in tomatoes.

The mechanisms of tolerance and resistance to TMV infection are not understood, but an antiviral compound has been identified in tomato plants systemically infected with TMV (Chadha and MacNeil, 1969), although its importance in relation to resistance has not been established. Grafting experiments between TMV-resistant and susceptible tomato genotypes have shown that the reaction to TMV of a tolerant or immune scion can be changed by grafting it on to a stock of certain susceptible genotypes (Arroyo and Selman, 1977). These results also indicate that translocatable compounds may be implicated in resistance to TMV.

Several forms of resistance to TMV are apparently race-specific and the presence of resistance-breaking strains of TMV has been demonstrated in many countries (Wilkinson, 1969; MacNeill and Fletcher, 1971; Pelham, 1972; Fletcher and Butler, 1975).

Tolerance and hypersensitivity to TMV are associated with poor fruit set, and plants that are homozygous for the resistance genes involved are usually unproductive (Laterrot, 1973). Nevertheless, many TMV-resistant tomato

varieties have been released, including Virocross and Supercross, which are heterozygous for the resistance gene *TM-1* that controls tolerance to TMV. However, a strain of TMV which causes severe symptoms on these varieties has become prevalent in the UK wherever they have been grown, and this tolerance is clearly race-specific (Pelham, Fletcher and Hawkins, 1970).

CURLY TOP

Curly top virus (CTV) can cause very serious damage to tomatoes in some western areas of the USA. This virus is transmitted by several species of leafhoppers, of which *Circulifer tenellus* (the beet leafhopper) is the most important. CTV has a very wide host range and can infect many common weed species as well as several important agricultural and horticultural crop plants, including tomatoes, sugar beet and *Phaseolus* beans. Much effort has been devoted to breeding CTV-resistant varieties of tomatoes, because other control methods have not been very successful.

Randell (1966) distinguished several different types of reaction to curly top in tomatoes, including tolerance, resistance to virus multiplication, and extreme resistance. Two main types of extreme resistance were identified, one being effective against certain strains only. Plants that express another kind of resistance show very severe curly top symptoms at first after inoculation with CTV, but soon recover from that disease. A similar recovery from curly top was found in some tomato lines by Benda and Bennett (1967), who also showed that plants which had been inoculated as seedlings usually expressed only mild symptoms of curly top when they grew older. This resistance is race-specific, because the variety VR Moscow showed a good recovery when infected with some CTV strains, but not with others.

Thomas and Martin (1969; 1971a) discovered a high level of resistance to CTV infection (disease escape) in some breeding lines that had been developed in the USA. However, the plants that became infected showed severe curly top symptoms and did not always recover from the disease. Recovery from curly top is, therefore, not always associated with resistance to virus infection. Disease escape seems to involve at least two separate components, which are resistance to the establishment of CTV in the host plant (Thomas and Martin, 1971a), and the preference of the leaf hopper vectors for certain tomato lines and varieties (Thomas and Martin, 1971b). Some of the resistant lines expressed either resistance to virus establishment or vector non-preference, and these two resistance components seem to be independently inherited. Martin (1969) reported that resistance to CTV infection is controlled in a breeding line, CVF4, by one incompletely dominant gene, together with at least one other dominant modifying gene.

Resistance to CTV has also been found in certain wild species of *Solanum* and *Lycopersicon*, including *S. pennellii* (Martin, 1962), *L. peruvianum* and *L. pimpinellifolium* (Martin and Thomas, 1969). A breeding line that had been derived from crosses involving *L. peruvianum* vars. *dentatum* and *humifusum* and *L. pimpinellifolium*, has shown excellent resistance in severe natural field epidemics of curly top. The inheritance of resistance in this line is complex and quantitative (Martin and Thomas, 1969). Line C5, which has been released

to breeders as a source of curly top resistance, has several desirable characteristics other than virus resistance, for example uniformity of ripening, earliness, resistance to cracking of the fruit and suitability for machine harvesting. However, some of the resistance to curly top shown by the resistant parent, *L. peruvianum* var. *dentatum*, has been lost during the breeding of lines CVF4 and C5 (Moser and Cannon, 1972); it may, therefore, be possible to develop commercially acceptable varieties with even higher levels of resistance, from such parents.

Although there are many strains of CTV, differing in host range and effects on different host plants (Thomas, 1969), resistance-breaking strains of CTV do not seem to have been a problem in the breeding or exploitation of curly top-resistant tomato varieties. However, some resistant lines have expressed greater resistance to CTV in some areas than others (Martin, 1963) and the existence of resistance-breaking strains of CTV cannot be ruled out.

SPOTTED WILT

Tomato spotted wilt virus (TSWV) is common throughout temperate and subtropical regions of the world. The disease causes severe stunting, chlorosis and necrosis in susceptible tomato varieties and this leads to reduced yield and low-quality fruit. Although TSWV is sap-transmissible, it is usually transmitted in the field by various species of thrips, particularly *Thrips tabaci*. The virus must be acquired by the vector in the larval stage, but it can be transmitted by adults only.

Breeding for resistance has been carried out in several countries, notably the USA and Australia. Holmes (1948) identified a type of resistance based primarily on a hypersensitive reaction; this was later shown to be race-specific. Strains of TSWV, discovered in New Jersey, overcame the resistance of the variety Pearl Harbor, and of related varieties that had been bred in Hawaii. If this New Jersey strain had been introduced into Hawaii, the susceptible local varieties might have been seriously damaged by TSWV. There is, therefore, a danger in introducing new virus strains into an isolated area such as Hawaii, even when other strains of that virus are already present (Holmes, 1955). Fortunately, resistance to the New Jersey strain of TSWV was found in the variety Rey de los Tempranos, this resistance apparently being controlled by a single recessive gene.

Three independently inherited resistance genes and two dominant alleles each control resistance to one of five groups of TSWV strains in tomatoes (Finlay, 1953). One line of *Lycopersicon pimpinellifolium* was resistant to all strains of TSWV with which it was inoculated, except the TB₂ strain; Rey de los Tempranos expressed a similar degree of resistance but had better agronomic characteristics. Finlay (1953) found that Pearl Harbor is resistant to strain TB₂, the resistance being controlled by the allele SW₁^b. *L. peruvianum* shows extreme resistance to TSWV in the field, and may prove to be a particularly useful source of resistance in future breeding programmes (Hutton and Peak, 1949).

CONCLUSIONS

Good sources of resistance to tomato mosaic, curly top and spotted wilt have been identified and used successfully in breeding programmes. Although some forms of resistance to these viruses were found in existing cultivated tomatoes,

more effective types of resistance to all three viruses have been derived from wild species of *Lycopersicon*, particularly *L. peruvianum* and *L. pimpinellifolium*, and from *Solanum pennellii*. Much of the resistance which has been used by breeders is race-specific, however, and great care will have to be taken to use the available resistance genes to the greatest effect. Where genes controlling different forms of resistance (i.e. extreme resistance, hypersensitivity, resistance to virus infection and virus tolerance) are available, it is probably desirable to combine as many of these as possible in the same variety, as suggested by Pelham *et al.* (1970) for TMV resistance.

Raspberries

Raspberries, like many vegetatively propagated crops, are subject to attack by a very large number of viruses. These viruses can conveniently be considered in two main groups, aphid- and nematode-transmitted. Aphid-transmitted viruses include raspberry mosaic and vein chlorosis viruses; these often occur together in the same plant and cause rather similar types of symptoms. Nematode-transmitted viruses include tomato ringspot, raspberry ringspot and arabis mosaic viruses. Varieties differ considerably in resistance to both groups, and resistant varieties are an important means of control.

APHID-TRANSMITTED VIRUSES

Aphid-transmitted viruses can cause serious damage to raspberries wherever susceptible varieties are grown. Slate, Braun and Mundinger (1953) were among the first to report varietal differences in resistance to the viruses that cause red and yellow mosaic diseases. For example, bushes of Newburgh remained apparently virus-free under conditions in which more susceptible varieties, such as Cuthbert, became badly infected. This resistance in Newburgh seems to be controlled by major genes, this variety being heterozygous for the resistance genes. Newburgh has also shown good resistance for many years, in the UK, to a complex of aphid-transmitted viruses (Knight and Keep, 1958), expressing both resistance to virus infection and virus tolerance. Newburgh is also resistant, or tolerant, to vein chlorosis virus (Cadman and Wood, 1951). Several varieties, including Newburgh, Malling Promise, Malling Landmark, September and Muskoka have shown good resistance to mosaic diseases in the USSR (Kichina, 1972). Freeman and Stace-Smith (1970) confirmed in Canada that the varieties Newburgh and Willamette show some tolerance to raspberry mosaic, which is caused by a complex of two viruses, one of which is heat-labile and the other heat-stable. Although Newburgh supported large populations of the aphid vector, *Amphorophora agathonica*, Willamette expressed resistance to this aphid; the rate and extent of spread of mosaic diseases in raspberries is therefore related, to some degree, to heritable differences in susceptibility to aphids (*see* page 365). The susceptibility of Newburgh to aphids may help to explain the high incidence of raspberry mosaic in some crops of this variety in British Columbia (Converse, Stace-Smith and Johnson, 1970). A recent reduction in the incidence of mosaic disease in north-western States of the USA has been attributed to increased popularity of aphid-resistant varieties including Willamette, and to a

decreased acreage of aphid-susceptible varieties such as Newburgh and Cuthbert. Reports of resistance in Newburgh have probably arisen mainly from areas in which populations of *Amphorophora agathonica* are low, or where other *Amphorophora* species, to which Newburgh is not unduly susceptible, are the main virus vectors.

Although virus-resistant lines, for example derivatives of Mallings Exploit (Jennings, 1963), are being used in many breeding programmes, 'virus-avoidance' through aphid resistance is often a preferred objective (Keep and Briggs, 1971). Zivanović (1974) tested 16 raspberry varieties for aphid resistance and found that resistance to one species of aphid is not always associated with resistance to other species. However, several varieties including Mallings Exploit, Wädenswil Red and Gradina are resistant to both these species, which are the most important aphid vectors of raspberry viruses in Europe. Ourecky (1976) found that resistance to *Amphorophora agathonica* and *Aphis rubicola*, the two main American aphid vectors of raspberry viruses, is controlled by different genes. Lloyd George is a major source of resistance to *A. agathonica*, and several varieties with resistance to this aphid species have been developed from it, including Canby and Citadel. The only red raspberry variety found by Ourecky (1976) to be resistant to *A. rubicola* was Williamette. However, a purple raspberry variety, New York 632, is apparently immune to both *A. agathonica* and *A. rubicola*. Resistance to aphids in raspberries is controlled oligogenically and, although it may be race-specific (see page 367), aphid-resistant raspberry varieties can give an excellent control of mosaic diseases in the field (Jones, 1976).

NEMATODE-TRANSMITTED VIRUSES

Varietal differences in resistance to *Arabidopsis* mosaic virus, raspberry ringspot virus and tomato blackring virus, each of which is transmitted from root to root by free-living nematodes, have been reported by Jennings (1964). Resistance to each virus is inherited as a dominant character, which is controlled by at least two genes. The genes controlling resistance to these viruses are different but are loosely linked. Taylor, Thomas and Converse (1966) reported that Mallings Promise, Lloyd George and Norfolk Grant, which had previously been immune to *Arabidopsis* mosaic, often became infected with this virus in Scotland. The reason for the apparent breakdown of the resistance of these varieties is not understood but a new virus strain, capable of overcoming the resistance, may be responsible.

Varietal differences in resistance to tomato ringspot virus have also been observed in raspberries. In Canada, virus infection reduced the vigour and yield of Fairview but had little effect on Newburgh, Sumner and Williamette (Freeman and Stace-Smith, 1968). In the UK, Glen Clova and Meeker were less damaged by this virus than eight other varieties tested (Scottish Horticultural Research Institute, 1974). Freeman, Stace-Smith and Daubeney (1975) compared the effects of tomato ringspot virus on ten raspberry varieties and found that growth was retarded by infection in all these varieties, but least in Meeker and Puyallup; the most affected varieties were Avon and Lloyd George, in which drupelet set was significantly reduced by infection. In other experiments, virus infections had no effect on drupelet set in several varieties including Canby, Carnival, Glen Clova, Mallings Jewel and Meeker (Daubeney, Freeman and Stace-Smith, 1975).

Bananas

Virus diseases are probably not a major factor limiting the yield of bananas in Africa or the western hemisphere, although plantations often become infected with cucumber mosaic virus. In the East, however, two virus diseases, bunchy top and abaca mosaic, can cause serious damage. Resistant varieties contribute to the control of these diseases (Meredith, 1970; Stover, 1972).

BUNCHY TOP

Bunchy top, which is endemic in South-east Asia, is transmitted by an aphid, *Pentalonia migranervosa*, in the persistent manner (see page 215). Symptoms of the disease include stunting and leaf chlorosis, and the yield of fruit can be greatly reduced on infected trees.

In Australia, Magee (1948) found considerable differences in resistance to virus infection between varieties; for example Gros Michel was more resistant than Cavendish varieties. Plants of several varieties from Fiji, Australia, Ceylon, Egypt and North Borneo, were each infested with 20 viruliferous larvae of *P. migranervosa* in his experiments. Under these conditions, plants of Gros Michel escaped infection with bunchy top. However, this resistance to virus infection was effective only when small numbers of viruliferous aphids were used for the inoculation. Although Gros Michel is not well adapted to Australian conditions, Magee (1948) considered that this variety would be a very useful source of resistance in Australian breeding programmes. Other varieties, including Veimama from Fiji, have also shown some recovery from bunchy top but they have, nevertheless, suffered large reductions in yield of fruit when infected. These are therefore much less satisfactory sources of resistance than Gros Michel.

Bernado and Umali (1956), working in the Philippines, found extreme resistance to bunchy top in a variety Pacol (*M. balbisiana*) and the resistance of another variety, Canton (*Musa textilis* × *M. balbisiana*), was also promising. Other hybrids between *M. textilis* and *M. balbisiana* showed extreme resistance to both bunchy top and abaca mosaic. Certain *Musa* clones with ABB genomes (the A genome being derived from *M. acuminata* and the B genomes from *M. balbisiana*) are resistant to infection with bunchy top virus in the field (Vakili, 1969). A triploid hybrid, Pisang Raja, with the genomes AAB, has expressed some degree of tolerance to bunchy top.

ABACA MOSAIC

Abaca mosaic virus is a major disease of bananas in the Philippines. It is transmitted by several species of aphids, including *Rhopalosiphum maidis* and *Aphis gossypii*, in the non-persistent, stylet-borne manner. All commercial varieties of *Musa textilis* are susceptible to abaca mosaic but *M. balbisiana*, *M. banksii* and *M. ornata* show extreme resistance to abaca mosaic virus (Bernado and Umali, 1956). Good resistance to abaca mosaic virus is also shown by the variety Canton, derived from a cross between *M. textilis* and *M. balbisiana*, and some hybrids are resistant both to abaca mosaic virus and bunchy top virus. However, in spite of these potentially useful sources of resistance to abaca mosaic virus, no resistant varieties have yet become widely grown.

Cotton

LEAF CURL

Leaf curl disease was first reported in 1912 on native cotton (*Gossypium barbadense*) in Nigeria, but it was not until 1926 that it was shown to be caused by a whitefly-transmitted virus. The disease is also found in certain other parts of Africa including the Sudan, where it is a serious disease in some areas.

The vectors are several species of *Bemisia*, of which the most important is *B. tabaci*. Severely infected susceptible plants of *G. barbadense* (Egyptian cotton) have a characteristic twisted appearance, the petioles and fruiting branches being particularly affected. The main stems of infected plants are often greatly elongated, leading to tall and spindly growth. In American cotton, however, infection leads to a shortening of the internodes and a bunchy-top type of growth. *G. hirsutum*, the species to which most cultivated cottons belong, is generally much more resistant to leaf curl than *G. barbadense*; this may help to explain the rather limited distribution of leaf curl virus (Tarr, 1951).

Much attention has been paid to the breeding of the leaf curl-resistant cotton varieties, particularly in the Sudan and in Nigeria. Although Egyptian and Sea Island cotton varieties are generally susceptible to leaf curl, resistant cotton strains were selected from an Egyptian variety, Sakel, as long ago as 1920. This early work was extended by Lambert (1938), who developed two varieties of cotton, X1530 and X1730, from plants selected for resistance from a field of Sakel in 1924. These were multiplied and subsequent tests showed them to be highly resistant to leaf curl. The effectiveness of this early selection programme is demonstrated by the fact that derivatives of X1730 were the most resistant to leaf curl of varieties tested 30 years later by Giha and Nour (1969). Selection continued in the Sudan for improved resistance to leaf curl with the particular objective of developing resistant strains of high quality.

One of the main disadvantages of these selections was the relatively poor lint quality of the resistant cottons. However, Siddig (1968) in the Sudan developed promising varieties from an original cross between the high-quality variety, Sakel, and a lower-quality but leaf curl-resistant variety, Lambert.

The nature of resistance to leaf curl is obscure. Resistant strains usually exhibit only mild symptoms and the virus can be transmitted from resistant plants only with difficulty, plants becoming infectious only when symptoms are clearly visible. This implies that the resistance of cottons such as X1730 involves both virus tolerance and resistance to virus multiplication. Resistance to the vectors may also be implicated. It was first reported in the 1930s that resistance to leaf curl in Nigerian cottons is sometimes associated with hairiness of the leaves, which in turn is associated with resistance to the whitefly vectors; this type of resistance to the vectors can be overcome by heavy infestations of insects. In the Sudan, however, leaf curl-resistant varieties of cotton are not usually hairy, and resistance to *Bemisia* spp. may not be an important factor in the leaf curl resistance of Sakel and its derivatives.

Knight (1948) reported that Sakel does not seem to possess any major genes controlling leaf curl resistance, although resistance can easily be built up by repeated cycles of selection. Siddig (1970), however, concluded from crosses between two susceptible and two resistant varieties, that resistance is controlled by a single dominant major gene, although modifying minor genes are also

present. These conflicting results may be partly due to the occurrence in the Sudan of two quite different types of leaf curl symptoms, which may be caused by two distinct viruses, both of which are transmitted by *Bemisia tabaci* (Bink, 1975; Ebbels, 1976). The first type of symptom is important only in certain *G. barbadense* varieties, particularly Isham and Sakel; the second is important in both *G. barbadense* and *G. hirsutum*. Siddig (1970) suggested that one of the viruses, which have differing distributions in the Sudan, causes thickening of the main veins of the lamina, whereas the other virus causes thickening of the small veins. Further work is needed to determine whether these virus isolates are different strains of leaf curl virus or distinct viruses. Apart from this possibility of independently inherited resistance to two viruses, there is no evidence that resistance to leaf curl is race-specific; resistant cotton varieties derived from Lambert's selections have remained resistant in the Gezira area of the Sudan since 1930.

Barley

BARLEY YELLOW DWARF

Barley yellow dwarf virus (BYDV) is a common pathogen of wheat, barley, oats and certain grains in many parts of the world, including North America, New Zealand and Europe. Barley yellow dwarf is particularly widespread and damaging in North America (Rochow, 1961; Gill, 1975). Yield losses of more than 70 per cent have occurred in susceptible barley varieties as a result of infection (Watson and Mulligan, 1960; Doodson and Saunders, 1970).

BYDV is not easily transmitted by mechanical inoculation and is invariably transmitted in the field by aphids in the persistent manner. Although the spread of BYDV can be reduced and delayed by controlling the aphid vectors with insecticides, control by resistant varieties would be easier and cheaper. Programmes of breeding for resistance to BYDV have, therefore, been started in several countries. Suneson and Ramage (1957) were among the first workers to recognize that some barley varieties, for example Rojo, were less damaged by BYDV infection than others; this tolerance is controlled by a recessive gene (*yd₁*). Schaller, Rasmusson and Qualset (1963) tested more than 7000 barley varieties for tolerance to BYDV and found that about 100, mainly from Ethiopia, were tolerant. Further work has shown that resistance to BYDV is non-randomly distributed in the centre of origin of *Hordeum vulgare* in Ethiopia (Qualset, 1975). Thirteen per cent of the Ethiopian and 40 per cent of the Sudanese barleys that were tested by Qualset and Schaller (1969) were resistant to BYDV. Some Ethiopian varieties have been tolerant to BYDV in some parts of the USA but not in others (Arny and Jedlinski, 1966), suggesting that different virus strains occur in different places and that tolerance to BYDV is race-specific. The existence of distinct strains of BYDV has been demonstrated by several workers, including Rochow (1961), and Jones and Catherall (1970a); specific interactions between individual host genotypes and particular virus strains have also been demonstrated (Gill and Buchannon, 1972). Resistance-breaking virus strains are, therefore, a potential threat to BYDV-resistant barley varieties.

Schaller *et al.* (1963), and Damsteegt and Bruehl (1964), showed that tolerance in most Ethiopian barleys is controlled by an incompletely dominant gene,

*Yd*₂. Later work by Catherall, Jones and Hayes (1970) showed that, in a barley line CI 1237, resistance gene *Yd*₂ is dominant in one environment and recessive in another. Plants which carry the *Yd*₂ gene express greater tolerance when they are growing rapidly than when growth is slow; this is so whether the growth rate of the host plant is determined by the genotype of the host or by the environment (Jones and Catherall, 1970b). The expression of tolerance in adult plants is also most pronounced when they have been inoculated with BYDV as seedlings (Comeau and St.-Pierre, 1975). The expression and dominance of tolerance derived from Ethiopian barleys therefore varies in different host genotypes, at different growth stages of the plant and in different environments.

Similar methods of screening for tolerance to BYDV seem to have been used by different workers. In glasshouse tests, Catherall and Hayes (1966) inoculated seedlings with five to ten viruliferous aphids, which were allowed to feed on the seedlings for two days. The aphids were then killed with an insecticide, after which the plants were grown on for disease assessment. The severity of BYDV symptoms, particularly leaf yellowing and stunting, was assessed by eye six to eight weeks after inoculation. The number of viruliferous aphids used to inoculate each plant is of crucial importance, because more aphids per plant are needed to produce the same amount of disease in older plants than in seedlings, and in some barley genotypes than in others at the same growth stage (Smith, 1967).

Field inoculations of BYDV are usually carried out by distributing pieces of BYDV-infected leaves carrying aphid vectors among the plants to be inoculated; as the pieces of infected leaves wilt, the aphids crawl from them on to the field plants, to which they transmit the virus. These aphids are generally killed two to four days later by spraying the plants with an appropriate insecticide (Catherall and Hayes, 1966; Jenkins, 1966). The severity of symptoms on the leaves, and the degree of stunting caused by BYDV, are assessed throughout the growing season, and grain yields from virus-infected and virus-free plots of each variety are compared. Field inoculations at an early growth stage have been found to reduce the grain yield of susceptible barley varieties (e.g. Proctor) by about 93 per cent, but do not significantly reduce the yields of two BYDV-tolerant Ethiopian barleys (Jenkins, 1966).

Yield tests have also been carried out in the glasshouse. Gill, Buchannon and Westdal (1969) reported that Herta lost 94 per cent of its potential grain yield because of BYDV infection in glasshouse tests, whereas the yield of Husky was reduced by only 55 per cent. These tests confirmed that the severity of chlorosis shown by infected leaves is an unreliable guide to the loss of grain yield that results from virus infection. There is not always a close agreement between the results of field and glasshouse experiments; of 14 barley varieties that were found to be tolerant to BYDV in glasshouse tests in Wales, only two were tolerant in field tests (Catherall and Hayes, 1966). These results confirm that the expression of tolerance to BYDV can vary greatly in different environments.

Although very little is understood about the mechanisms that are involved in resistance to BYDV, it is generally agreed that virus tolerance is the most important resistance component. Orlob and Arny (1961) have shown that the effects of BYDV infection, which include an increased respiration rate, the accumulation of starch and reducing sugars in the leaves and a decreased catalase activity, are considerably less in tolerant plants than in susceptible plants. Resistance to virus multiplication is also probably implicated, because Catherall

and Hayes (1966) showed that aphids can acquire and transmit BYDV less readily from tolerant Ethiopian barleys than from more susceptible varieties, presumably because resistant varieties contain less virus. The gene Yd_2 may therefore control both virus tolerance and resistance to virus multiplication. Some barley lines, for example CI 3906-1, show a tendency to escape BYDV infection (Munthe, 1975), this resistance to virus infection being controlled by a small number of recessive genes that differ from those controlling virus tolerance. Tanna (1971) had also found that a tendency to escape disease is inherited independently of virus tolerance; some of the resistant barleys which he examined were resistant to BYDV infection and others were tolerant.

New sources of resistance to BYDV are urgently needed because, as Schaller (1977) has pointed out, plant breeders throughout the world have used the same resistance gene (Yd_2). There is, consequently, a danger of a serious and widespread breakdown in resistance if there is a shift in virulence of BYDV leading to the development of resistance-breaking virus strains.

References

The references cited in this chapter, together with those for Chapters 5, 6, 7 and 8 are listed in References – Part III, pages 267–290.

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PESTS – SOME GENERAL CONSIDERATIONS

Economic Importance of Pest Attack

Insect pests have been estimated by Cramer (1967) to cause an annual world crop loss of nearly 14 per cent of the potential yield. The calculated losses for individual crops vary from 5.0 per cent in wheat to a staggering 26.7 per cent in rice. Yield losses are considerably more than 5 per cent in most of the major agricultural crops of the world, in spite of the widespread use of insecticides and other control measures. Insect damage is usually much more extensive in tropical and subtropical crops than in those of temperate regions, because high temperatures tend to increase insect activity and reproductive potential. The economic importance of insect pests to agriculture is even greater if one includes damage by insects to stored products, and the effects of insect vectors of viruses and mycoplasmas, which cause very great yield losses in crop plants.

Cramer's estimates of pest damage to crop plants are restricted to insect pests and take no account of the effects of other injurious animals such as nematodes, slugs and snails, mites and vertebrates. Plant parasitic nematodes, which include free-living ectoparasites, and free-living and cyst-forming endoparasites, can cause extensive damage in many crops. Although slugs and snails are not among the most damaging pests on a world-wide basis, they can cause severe crop losses locally where wet or humid conditions prevail. Mites can seriously damage crop plants by sucking sap from host plants and also by transmitting virus diseases.

Some of the most damaging crop pests are birds and small mammals (Walker, 1975). For example, the African Quelea or weaver bird causes enormous yield losses in maize, sorghum and other crops in the semi-arid parts of Africa, and is undoubtedly one of the world's most destructive crop pests. Rats can devastate crops and, in addition, can consume or spoil vast amounts of stored products. Recent reports from Senegal, Africa, show that invasions of the countryside by millions of rats have threatened about 150000 people with starvation. Rabbits, each eating up to 0.5 kg of vegetation every day, can also cause very great damage to crops.

There are so many important non-insect animal pests of crop plants that it would be surprising if they did not cause damage at least as great as that caused by insects. If this assumption is correct, the annual world crop losses caused by all pests combined probably exceeds 25 per cent of the world's total potential crop production.

Agronomic and Chemical Control Methods

For a detailed account of pest damage and pest control the reader is referred to two of several excellent textbooks on these subjects, for example those by Jones and Jones (1974), and Woods (1974).

The control of invertebrate pests of growing crops has been based on three main types of measures: cultural methods, pesticides and biological control, which includes the use of resistant varieties. Cultural methods include the removal of alternate hosts between successive susceptible crops, manipulating the sowing date to escape or reduce damage, crop rotation, and the use of appropriate soil cultivation techniques. Pesticides, which can be applied as foliar sprays, seed dressings or granules added to the soil, can give a very good control of most insect pests if they are applied correctly and at the most suitable time. It is, however, often difficult to apply pesticides at the appropriate time, and they can be dangerous to use, can pollute the environment and are expensive. In addition, forms of many pests which are resistant to pesticides have become widely distributed, so that effective chemical control cannot easily be achieved in the field or glasshouse. Planned biological control of invertebrate pests using natural enemies, both predators and parasites, has been successful in certain localized situations such as in glasshouses or on isolated islands, but is not yet of any general significance. Biological control by resistant varieties is of much greater importance and has made a very significant contribution to the control of many invertebrate pests.

Vertebrate pests, mostly birds and small mammals, are notoriously difficult to control. The main control methods have been scaring, trapping, shooting and poisoning, although biological control has been effectively used in the control of some vertebrate pests, for example, rabbits by myxomatosis virus. Resistant varieties have not been important in reducing crop damage by vertebrates.

Some Characteristics of the Main Groups of Animal Pests

NEMATODES

Nematodes, which are often referred to as 'eelworms', are threadlike roundworms which are hardly visible to the naked eye. They are among the most abundant groups of animals, both in number of different species and in number of individuals. A bucketful of earth may contain more than one million individual nematodes but, fortunately, most of these are harmless or even beneficial to crop plants. Many of the soil-inhabiting nematodes help, directly or indirectly, to maintain soil fertility.

Plant parasitic nematodes can be classified into three main groups according to their life history and behaviour.

Free-living ectoparasites

These nematodes move in the air spaces between soil particles and feed on the roots of a wide range of host plants. Several species, particularly those which are common in light, sandy soils, are vectors of certain plant viruses. These

nematodes can be partially controlled by improving the condition and fertility of the soil, for example by incorporating organic matter and nitrogenous fertilizers into infested soil. Chemical treatments, such as fumigation with dichloropropene or dichloropropane, or the incorporation of aldicarb granules into the soil, have been effective but are too expensive to use on most field crops. Most species of ectoparasitic nematodes have such wide host ranges that the development of varieties that are resistant to them may seem unlikely.

Free-living endoparasites

These include the root-knot eelworms and the stem eelworms, and are destructive pests of several important crops. They feed inside plant tissues, although they are commonly found in the soil near their host plants.

Root-knot eelworms, which are particularly damaging to tropical and sub-tropical crops, cause galls on the roots of host plants. Inside these swellings are microscopic, pear-shaped female nematodes, each of which contains several hundred eggs. Most larvae, which hatch from the eggs and burrow into the host tissues, start to reproduce four to five weeks later. However, some larvae emerge from the root into the soil, where they can survive, without feeding, for many months. These free-living larvae are the main means by which root-knot nematodes can survive between one susceptible crop and the next. Infestations of root-knot nematodes can be controlled by steam-sterilization of the soil, by soil fumigation or by applications of suitable nematicides. Resistant varieties of several important agricultural and horticultural crops have been developed, and these have contributed significantly to the control of root-knot nematodes.

Stem eelworms, a group of nematodes which includes *Ditylenchus dipsaci*, an important pest of several crop species, also live inside plant tissues, where both males and females occur in profusion. Microscopic larvae hatch from eggs and may grow and reproduce to such an extent that their feeding kills the host-plant tissues which contain them. Adults can migrate through the soil from the roots of one plant to those of another, and spread within a crop occurs in this way. When host-plant tissues are killed, or so badly damaged that food is no longer available to the nematodes, masses of the larger larvae ooze out of the host and become dormant in the soil; they can live in this state for many years without further food. Control is usually effected by destroying residues of infested plants and treating infested soil with appropriate chemicals. Nematodes such as *Ditylenchus dipsaci* have very wide host ranges and the possibility of developing resistant varieties may seem remote. However, resistant varieties of clover (see page 362) and oats (see page 333) have been produced, and these have made a significant contribution to the control of *D. dipsaci* in several countries.

Cyst nematodes

These nematodes lie dormant in the soil as cysts, which are the dead remains of adult female eelworms containing very large numbers of eggs inside which are larvae. The young larvae escape from the cysts, invade the roots of host plants and feed on the cortical cells, moulting as they enlarge. Female larvae become swollen and the plant tissues around them break down, so that the swollen bodies protrude into the soil. Adult males break out into the soil and mating

occurs on the outside surface of the root. Development of the eggs occurs within the body of the female, which becomes a cyst. During the growing season of the host, the eggs inside the cyst hatch to produce larvae, which can either invade the tissues of the same host plant or migrate through the soil to infest the roots of other plants. As the host matures, the cysts become resting bodies containing eggs which usually hatch only in the presence of specific diffusates from the roots of potential host plants.

Among the fifteen or so species of true cyst-forming nematodes are the potato cyst eelworms, *Globodera rostochiensis* and *G. pallida*, the sugar beet root eelworm, *Heterodera schachtii*, and the cereal cyst nematode, *H. avenae*, all of which are widespread and damaging crop pests. Although nematicides can give a good control of cyst nematodes, crop rotation is also effective and may be a cheaper alternative; appropriate crop rotations of susceptible and resistant crops are often required by legislation.

Resistant varieties have played an important part in controlling the potato cyst and cereal cyst eelworms. Several potato varieties, including Maris Piper and Pentland Javelin in the UK, are resistant to the most widely distributed pathotype of *G. rostochiensis* (see page 356). The larvae are attracted to the roots of resistant plants and invade them as readily as those of susceptible plants, but only males complete their life cycle on resistant plants and no females are produced. The main effect of resistance is, therefore, to decrease the nematode population in the soil for succeeding crops of potatoes, because no new cysts are produced on resistant varieties. Infested soil thus becomes progressively depleted with each successive crop of resistant potato varieties. This resistance is particularly effective when it is used in conjunction with nematicide treatment and an adequate rotation with non-host crops. Attempts are now being made to breed varieties with equally effective resistance to *G. pallida* and other pathotypes of *G. rostochiensis*.

Varieties of spring barley, for example Sabarlis, which are resistant to two pathotypes (biotypes) of *Heterodera avenae*, have recently been marketed in the UK (see page 331). These varieties significantly outyield susceptible varieties on infested soils and also reduce the eelworm populations in the soil. In spite of similar efforts to develop varieties of sugar beet that are resistant to the beet cyst nematode, no resistant varieties have so far been produced (see page 340).

Nematode infestation of crops usually occurs in patches because eelworms are unable to travel in the soil far from the initial scattered infestations. Dissemination of nematodes is usually by means of infested soil or infested plant material, particularly propagating material such as potato tubers. This implies that, if effective precautions are taken to prevent accidental spread of nematodes through transport of infested soil or plant material, any resistance-breaking variants of nematodes which may arise are unlikely to spread rapidly from their point of origin. This situation contrasts strongly with that concerning air-borne pathogens or pests where resistance-breaking variants can be disseminated extremely quickly over a large area.

MOLLUSCS – SLUGS AND SNAILS

Over 100000 species of molluscs are known – more than any other animal Phylum except the Arthropoda – but fortunately very few of these species are

pests of crop plants and these are confined to one Class of Mollusca, the Gastropoda, which includes the slugs and snails. The other five Classes in the Phylum comprise mainly marine animals.

Snails and slugs are very susceptible to desiccation and they are therefore usually found in wet or damp environments. This requirement for dampness greatly restricts their distribution and limits their importance as agricultural pests. Nevertheless, they can cause local but severe damage to crops in many parts of the world. In the UK, for example, the extent of slug damage to wheat and potatoes has caused concern in recent years and more effective control measures are urgently being sought. Slugs can damage cereal crops in several ways. Under suitably moist conditions the embryo and endosperm of planted seeds can be eaten before germination, and this causes gappy crops. Leaves of cereal seedlings, which have just emerged from the soil, can be grazed by slugs, and the stems and leaves of older plants can also be damaged.

Potato tubers can be severely damaged by slugs, particularly on heavy soils in mild, wet growing seasons. Some potato varieties are known to be much more susceptible to slug attack than others and the planting of such varieties should be avoided in areas where slugs are a potential threat. In the UK, Stormont Enterprise, Pentland Dell and Majestic are reported to be moderately resistant, while King Edward, Desirée, Redskin and Maris Piper are particularly susceptible (Ministry of Agriculture, Fisheries and Food, 1973).

Chemical methods are widely used to control slugs, but cultural methods can also be helpful. Additional soil cultivation to break down large clods of earth, either before or after planting, can reduce slug attack, which is encouraged by the presence of large clods and a rough seedbed. The most widely used control method employs baits containing molluscicides, usually metaldehyde or methiocarb, mixed with bran. The poison bait must be scattered on to the soil at an appropriate time, and uniform application of bait over a large area can be difficult and expensive. Spraying the soil surface with copper sulphate or a metaldehyde solution before ploughing can also be effective.

Slugs and snails can feed on many types of vegetation; their damage is not, therefore, confined to particular types of crop plants. Nevertheless, they show preferences for soft, succulent plant material and even for particular genotypes of host plant. It has already been mentioned that certain potato varieties are more extensively attacked and damaged by slugs than others. The mechanisms which are responsible for these varietal differences have not been identified, but the differences show that breeding for resistance to slugs should be possible in many of those crop species which are most at risk.

ARTHROPODS – INSECTS AND MITES

The Phylum Arthropoda contains a greater number of species than any other group of animals. Three main Classes of the Phylum contain species which are major pests of agricultural crops; these Classes are the Myriapoda (millipedes), the Arachnida (mites) and the Insecta (insects). These Classes also contain many beneficial organisms.

Myriapoda

Centipedes are carnivorous and are usually beneficial to agriculture because they feed on many injurious insects. Millipedes are plant feeders and can attack most kinds of crop plants, but seedlings and young plants of sugar beet, peas, beans, carrots and potatoes are particularly susceptible to millipede damage.

Millipedes are considered to be serious pests of sugar beet in some northern parts of Europe and insecticides, including gamma-BHC and DDT, have been incorporated into the soil to control them. No serious attempts seem to have been made to identify resistance to millipedes in sugar beet or any other crop and, as long as they can be controlled by chemicals, it does not seem to be worth while to determine whether there are varietal differences to millipede attack.

Arachnida

Some species of mite are very serious pests of crop plants. The so-called 'two-spotted' or 'red spider' mite, *Tetranychus urticae*, has a very wide host range of field and glasshouse crops including beans, hops, cotton, soybean, tomatoes and cucumber. This mite feeds mainly on the underside of leaves, sucking the sap. Leaves of heavily infested plants become speckled and yellow or bronzed. The author has seen sugar beet crops in the Sacramento Valley of California which were so badly infested with the two-spotted mite that almost every plant had become very chlorotic and stunted.

Adult two-spotted mites are very small, with bodies seldom longer than 0.8 mm. Mating occurs on the host plants as the crops mature, and the females hibernate just beneath the soil surface or in other sheltered situations. In the Spring these females infest the young leaves of nearby host plants and lay eggs on them. After an incubation period of about two weeks the eggs hatch to immature mites. These pass through three larval stages before reaching maturity.

Other important mite pests include the blackcurrant gall mite (*Cecidophyopsis ribis*), and *Abacarus hystrix* which attacks ryegrass and other grasses. Each of these mites can cause crop damage in two quite different ways; first, by causing direct feeding damage and second, by transmitting plant viruses. The blackcurrant gall mite, for example, transmits the blackcurrant reversion virus from plant to plant. The resulting disease not only decreases the yield of fruit but also increases the suitability of blackcurrants for the gall mite vector.

Control of mites in the field usually involves the spraying of infested plants with chemicals; many acaricides have been developed for agricultural use, including a wide range of organophosphorus compounds. There have been many reports of failure to control mites by several individual acaricides when they had been used extensively for a few years. These failures were mostly caused by the development of acaricide-resistant strains of mites, and resistance to one chemical was usually associated with resistance to related compounds. Many mite populations have now developed resistance to most organophosphorus compounds.

The ephemeral control of mites by most acaricides, which has resulted from the pest's ability to produce resistant forms, has led to a search for other control methods. One approach has been to attempt biological control using species of predatory mites, for example *Phytoseilus* spp., which feed on injurious mites. *Phytoseilus* has given a good control of the two-spotted mite in glass-houses, provided that the predatory mites have been introduced at the most

suitable time, which is usually shortly after the pest has begun to multiply. The predatory mites have to be repeatedly re-introduced into infested crops and their management is sometimes too complex for use on a large scale in commercial glasshouses.

Varietal differences in resistance to attack by mites have been observed in many crop species. For example, some blackcurrant varieties are much less susceptible to gall mite (*Cecidophyopsis ribis*) than others (see Figure 10.1).



Figure 10.1 Shoots of resistant (left) and susceptible (right) blackcurrant seedlings after exposure to natural infestations of the blackcurrant gall mite (*Cecidophyopsis ribis*) for three years. (By courtesy of East Malling Research Station)

A dominant gene, *Ce*, which confers resistance to *C. ribis* has been successfully transferred from gooseberry to blackcurrant (Knight, 1977); this gene also confers resistance to blackcurrant reversion virus, of which *C. ribis* is the vector.

Resistance to the two-spotted mite (*Tetranychus urticae*) and the desert spider mite (*Tetranychus desertorum*) has been reported in cotton by Schuster

and Cherry (1975), and Schuster and Maxwell (1976). Varieties of soybean also differ in resistance to *T. urticae* (Bailey and Furr, 1975). Although relatively few attempts have been made to breed crop plants specifically for resistance to mites, it seems that there is ample scope for plant breeders to do so. However, the considerable ability of mites to develop acaricide-resistant variants suggests that resistance-breaking biotypes of mite pests could be a major problem in breeding for resistance.

Insecta

The millions of species which comprise the Insecta can be grouped into three main Subclasses; (1) Apterygota, (2) Exopterygota and (3) Endopterygota. These will each be considered briefly in turn.

Apterygota This Subclass consists of primitive, wingless insects, most of which are of no significant agricultural importance. Insects of the Order Collembola (springtails), which are very common in most soils, occasionally damage the roots of sugar beet seedlings (Baker and Dunning, 1975) but most springtails help to maintain soil fertility. Specific control measures are not usually necessary to control Collembola populations in the soil although soil insecticides have occasionally been used to reduce damage to sugar beet seedlings in parts of northern Europe. Breeding for resistance to springtails or other Collembola is unlikely to be worth while because of their relative unimportance as agricultural pests.

Exopterygota These are insects with wings developing outside the body and in which the nymphs are miniature adults. This Subclass contains many of the most damaging pests of agricultural crops, including locusts, aphids, leafhoppers and thrips. The nymphs, which may hatch directly from eggs or be born viviparously, grow to adulthood through a series of moulting stages called *instars*. The most important pests belong to three of the 16 Orders that are usually considered to comprise the Exopterygota; these are the Orthoptera (locusts), Thysanoptera (thrips) and Hemiptera (plant bugs, including aphids and leafhoppers).

Locusts are one of the most injurious of all crop pests; plagues of locusts can cause enormous damage to crops in India, Pakistan, the Middle East and parts of northern Africa. Locusts usually behave as solitary grasshoppers, which do not cause extensive crop losses. However, under certain conditions, particularly when the food supply in an area threatens to become exhausted, these insects congregate in large swarms containing thousands, or even millions, of locusts and mass migration occurs. These swarms can travel hundreds of miles in a period of two or three weeks and they leave a trail of destruction behind them, stripping entire fields of all vegetation with their powerful biting mouthparts in a very short time. They are omnivorous vegetarians and do not appear to have any special preferences for particular crops as they swarm.

Locusts are usually controlled by applying insecticides from aircraft to their breeding grounds. The possibilities of using natural enemies, predators and

parasites in controlling locusts are also being investigated. Work in South America nearly 40 years ago showed that the maize variety Armago is more resistant to a locust (*Schistocera gregaria*) than another variety, Piamontes; this resistance is controlled by a single, recessive gene (Horowitz and Marchioni, 1940). There seem to have been few reports of recent work on resistance to locusts in any crop plant but the possibilities should, perhaps, be studied in more detail. Unpalatability to locusts, or an ability to regenerate new growth quickly after a locust attack, might be particularly useful varietal characteristics of crops in areas which are especially at risk.

The Hemiptera (plant lice) are small insects with transparent wings, and mouth-parts which are modified for piercing plant tissues and sucking sap from them. Sap from host plants contains all the nutrients which an aphid requires for normal growth and reproduction. Although plant lice can overwinter as eggs, which are resistant to cold and desiccation, they usually reproduce parthenogenetically on their summer hosts. The larvae which are so produced are often born viviparously, so that they are immediately independent of their mothers.

Many Hemiptera are important crop pests which damage their host plants, both by extracting essential nutrients and water from them in the sap, and by transmitting many plant viruses. There is often a specific association between individual Hemipteran species and the viruses which they can transmit. For example, the aphid *Myzus persicae* is unable to transmit the curly top virus which can infect a wide range of host plants of this aphid, including sugar beet, tomatoes and *Phaseolus* beans; however, *M. persicae* is a very efficient vector of beet western yellows virus, which also infects sugar beet and many other species of crop plant. Conversely, the beet leafhopper (*Circulifer tenellus*) can transmit curly top virus from sugar beet to sugar beet, but is not a vector of beet western yellows virus. These relationships between plant viruses and their insect vectors are discussed in more detail in Chapter 8 (see page 215). The extent of damage to crop plants from viruses transmitted by aphids and leafhoppers is usually much greater than that attributable directly to their feeding activities. *Myzus persicae* can transmit more than 60 different plant viruses, many of which cause severe losses of yield in some of the most important temperate crops; this makes it, undoubtedly, one of the most damaging of all crop pests.

Control of Hemipteran pests usually involves applications of insecticides, either to the crop or to other plants on which the pest breeds. Contact insecticides are usually less effective than systemic compounds, which are sucked up by the insect with the sap during feeding. Organophosphorus insecticides have been very widely used to control aphids and leafhoppers and, if applied at the correct concentration and the most suitable time in appropriate weather conditions, can give an excellent control of these insects and of the viruses which they transmit. Unfortunately, insecticides can rarely be applied under ideal conditions and less than perfect control is usually achieved. In addition, forms of aphids that are resistant to organophosphorus insecticides have become widespread in many parts of the world. Other kinds of insecticides, for example carbamates, have subsequently been employed in these areas and at first gave a good control of aphids, but many of these have also become less effective because of the presence of insecticide-resistant forms.

Other methods of controlling Hemipteran pests are being developed, particularly those employing natural enemies of these pests (Way, 1974; Mackauer

and Way, 1976) and the breeding of insect-resistant varieties of crop plants. Breeding for resistance to aphids has been in progress for many years in several crops, including cereals (*see* page 327), potatoes (*see* page 360), sugar beet (*see* page 338), maize (*see* page 351) and brassicas (*see* page 369). Breeding for resistance to jassids and leafhoppers has been carried out in several crops, particularly cotton (*see* page 337) and rice (*see* page 344).

Endopterygota In the Endopterygota the wings develop *inside* the body and the young insects are larvae which are quite unlike the adult in form and behaviour. Metamorphosis is complete and is usually in three stages. (1) The egg hatches to produce an active, wingless larva which may be aquatic even where the adult is terrestrial. (2) The larva pupates when fully grown. This is a resting stage during which the insect changes from the larval to the adult form. (3) The adult, which is usually winged, emerges from the pupa and fulfils the functions of mating, reproduction and dispersal. In the Lepidoptera (butterflies and moths) the larva is usually called a caterpillar and the pupa a chrysalis; in other Groups, larvae are often known as grubs.

The Group Endopterygota comprises 11 Orders and contains both beneficial insects and pests. Important pests of crop plants are found in only four of these Orders; these are the Coleoptera (beetles and weevils), the Lepidoptera (butterflies and moths), the Hymenoptera (sawflies) and the Diptera (flies).

Many members of the Coleoptera feed on injurious insects, for example, ladybird beetles prey on aphids and help to control them. Others, such as weevils, are plant feeders and not only cause direct feeding damage to crop plants but also are vectors of several plant viruses. Larvae of weevils are soft, white, footless grubs which usually feed within the tissues of the host plant. Adult weevils, on the other hand, feed mainly on the outside of plants, using their powerful chewing mouthparts which are situated at the tip of their long snouts. Although weevils can sometimes cause severe damage in certain crop species, control measures are not usually necessary. Insecticides, applied either to the soil or to crop plants, can give an adequate control of weevil damage. Varieties of plants resistant to weevils have been developed in a few crop species; for example some varieties of cotton are resistant to the boll weevil, which can be an important pest in the USA (*see* page 336).

Resistance to the cereal leaf beetle (*Oulema melanopus*), which is a damaging pest of cereals in North America, has been demonstrated in wheat and barley (*see* page 329). Adult females of this pest lay fewer eggs on leaves of certain varieties, and relatively few larvae of *O. melanopus* survive on resistant plants.

The Order Lepidoptera contains many butterfly and moth species which are important pests. Adults, which usually have coloured, diaphanous wings, have sucking mouthparts, which are used for obtaining plant juices such as nectar from flowers, but do not pierce plant tissues; butterflies and moths are, therefore, harmless to crop plants but their larvae (caterpillars) are voracious plant feeders and, when numerous, can defoliate host plants very quickly.

Bollworms and earworms, which are the larvae of several species of moth, are important pests of cotton, sorghum and maize. Eggs are laid on the leaves and stems of host plants, and the caterpillars feed on the developing seeds and fruits. Although insecticides have been used to control these pests, resistant varieties have been very important, particularly in maize (*see* page 350).

Stem borers, which are larvae of several species of Lepidoptera, are major pests of rice and maize. The caterpillars burrow into the stem and leaf sheaths,

and heavy infestations can cause severe damage to the crop. Insecticides are the main control method, but there are good prospects of breeding for resistance to those pests in rice (see page 342) and maize (see page 350).

The many other Lepidopteran pests include larvae of the cabbage white butterfly, *Pieris brassicae*, which feed voraciously on plants of the cabbage family. Caterpillars of the Silver-Y moth (*Plusia gamma*) cause losses of yield in several species of crop plants. Although varietal differences in resistance to these pests have been noted in a number of crop species, few attempts to breed for resistance to them have been reported. Insecticides, and biological control using predators and parasites, have been the most widely used methods to reduce damage by these pests. If current control measures are found to be inadequate, there are clearly good prospects of breeding for resistance to *Pieris brassicae* and *Plusia gamma*. Dickson and Eckenrode (1975) have reported that *Pieris rapae* prefers to oviposit on certain types of cabbage, and this kind of 'pest escape' could be exploited in breeding for resistance. Long and dense hairs on the leaves and stems deter oviposition by many species of Lepidoptera; selection for increased pubescence in crop species which are particularly at risk from caterpillar damage might, therefore, form the basis of selection for resistance.

Relatively few of the 800 000 species of true flies (Order: Diptera) are serious pests of crop plants. The larvae of flies are legless maggots and, as with butterflies and moths, it is the larvae which can damage crop plants. Dipteran pests include the mangold-fly (*Pegomyia betae*), whose maggots mine inside the leaves of mangolds and sugar beet, and crane-flies (Tipulidae), whose larvae are known as leather-jackets and feed on the roots of grasses and many other crop plants. The larvae of the frit-fly (*Oscinella frit*) damage the shoots of wheat, barley, oats and maize; carrot-fly maggots feed on the roots of carrots. Insecticides are usually applied to control these pests, and resistant varieties of crop plants have not played an important part in reducing the damage which they cause. An exception is wheat, in which resistant varieties have played a very important part in controlling the Hessian fly (*Mayetiola destructor*), the larvae of which burrow into the stems of wheat, causing severe damage (see page 325).

Species of the Hymenoptera can conveniently be grouped into two Suborders: the Symphyta (sawflies), and the Apocrita (bees, wasps and ants). Sawflies are predominantly plant feeders, and several species are pests of crop plants. Unlike most other Hymenoptera, sawflies do not have constricted waists, and the ovipositor is modified into a saw. Adult females make a slit with this saw in the tissues of a host plant, into which they deposit eggs. These hatch to produce larvae, which resemble the caterpillars of the Lepidoptera. The larvae feed on the tissues of the host plant until they are fully grown, when they spin a cocoon in which they pupate. Adult females emerge from the pupae and seek new host plants in which to lay their eggs.

The wheat stem sawfly (*Cephus cinctus*) was a serious pest of wheat in North America until resistant varieties were grown extensively. Larvae of *C. cinctus* sever the vascular tissues of the host during feeding; this can seriously affect the filling of the grain by interfering with translocation of water and nutrients within the host. Solid-stemmed wheat varieties, which are much less severely damaged by sawfly attack than are other varieties, have been developed in the USA and have contributed significantly to the control of this pest (see page 328). Many other sawfly species attack crop plants, for example *Pteronous ribesii* infests gooseberry plants, but the host range of each species is usually very

narrow, indicating a very specific relationship between these pests and their host.

The Apocrita comprise both winged forms, such as bees and wasps, and also insects which have 'lost' their wings during the course of evolution, such as ants. Very few species in this Suborder are crop pests and most are beneficial to agriculture; many species prey on, or parasitize, injurious insects. Leaf-cutter ants are locally important pests in South America and can quickly defoliate entire crops. These pests are difficult to control, but treatment of their nests with insecticides has sometimes been effective. The insects carry pieces of leaf back to their nests, for use in culturing a particular species of fungus that is the staple diet of the ants. Presumably, if the leaves of crop plants were sprayed with an appropriate fungicide before they were cut and transported back to the nests, the fungi would not flourish and the ants would die of starvation. It is unlikely that varieties of crop plants will be developed that are resistant to leaf-cutting ants, because the ants are omnivorous plant feeders. For this reason, control of these ants will probably continue to be based on the use of pesticides.

VERTEBRATES

Some of the most damaging pests of crop plants are vertebrates, particularly certain birds and small mammals. For example, bush fowl, weaver birds, rats and mice probably cause the loss of nearly 50 per cent of the potential yield of pre- and post-harvest crop products in south-western Nigeria. The Quelea or weaver bird, which feeds in enormous flocks on maize, rice and sorghum, causes incalculable crop losses in parts of southern Africa (Murton and Westwood, 1976). Many other species of birds damage crops by eating either the leaves, stems or seeds of growing crops, or seeds that have been sown in the soil. Bird pests are notoriously difficult to control and the Quelea bird is no exception. Diverse methods of bird-scaring, including the use of explosives, have been unsuccessful, as have large-scale applications of poison to the breeding grounds.

There might, at first, seem to be little prospect of breeding varieties of crop plants for resistance to pests such as the Quelea bird. However, considerable progress has been made with the development of bird-resistant varieties of maize (see page 353) and sorghum (see page 377). Certain morphological characteristics, such as the presence of strong spines or awns, or thick, tough tissues, can contribute to bird resistance, as can the presence of distasteful substances.

The most important mammalian pests of crop plants are rodents, particularly rabbits, rats and mice. Rats and mice are very damaging to stored grain products because they consume vast amounts of food, and contaminate the remainder with their droppings and urine. A rabbit can eat immense quantities of leaves and roots, and large populations of grazing rabbits can cause enormous damage to growing crops.

Chemicals, applied as gases or in poison baits, are the usual means of controlling rats and mice. Warfarin, a compound which prevents the clotting of blood, has been a particularly valuable rodenticide. Unfortunately, many populations of rats and mice have developed resistance to warfarin, but new and promising rodenticides have been developed and are now being tested on a large scale. Biological control of rabbits by myxomatosis virus disease has greatly reduced populations of this pest in many parts of the world. However,

resistant populations of rabbits have become widespread and this pest is again causing severe damage to a wide range of crops.

Very little work has been carried out on breeding for resistance to mammalian pests, but the possibilities of resistance to the main pests should be explored. Rabbits show definite preferences for certain types of food plant, and some genotypes of crop species may be less palatable to rodents than others. Such differences might form the basis of selecting for resistance to mammalian pests.

Genetic Variability in Animal Pests

Many examples of the extent of genetic variation in pests of agricultural crops will be given in Chapter 11. For example, several pathotypes of the potato cyst nematodes are known to exist, each able to infest specific potato genotypes (see page 356). Insect pests are also very variable, and resistance-breaking biotypes of several aphid pests, including *Amphorophora idaei* (the raspberry aphid), have presented serious problems for the breeder. The great capacity of insects to produce new variants is illustrated by the rapid appearance of insecticide-resistant forms of many pests, and pesticide-resistant variants have developed in many other groups of animal pests, including mites and rodents.

Invertebrate and vertebrate pests seem, therefore, to have a great potential for genetic variation that can lead to the development of biotypes which are capable of attacking previously resistant varieties. In spite of this potential, however, resistance-breaking biotypes of pests have generally been less of a problem to breeders than have variants of fungal or bacterial plant pathogens. The reasons for this are not clear, but pest species may not have been exposed to the same intensive selection pressure to develop 'fit' resistance-breaking variants as have many plant pathogens. Nevertheless, pest-resistant varieties of many crop plants have been grown extensively for many years without the widespread occurrence of pest biotypes which can attack them.

It is important to distinguish between the following four main types of biotypes of pests in relation to resistant varieties of crop plants:

- (1) **True resistance-breaking biotypes**, which are variants that can attack previously resistant varieties. They are adapted to attack specific host plant resistances and there is, therefore, an interaction between the genotypes of host and pests (e.g. aphids in raspberries (see page 366) and cyst nematodes in potatoes (see page 357). In nematology, such biotypes are often called 'pathotypes' to distinguish them from other kinds of biotypes.
- (2) Some variants of a pest may be unusually vigorous with a high reproductive potential on **all** genotypes of a host species: Such biotypes colonize both resistant and susceptible host plants more effectively than other biotypes, but there is no specific adaptation to any particular host plant genotype (e.g. biotypes of the cabbage aphid, *Brevicoryne brassicae*, in rape (see page 369)).
- (3) Some variants of a pest may occur in certain geographical areas; they may or may not be true resistance-breaking biotypes. For example, some biotypes of the wheat Hessian fly seem to have different distributions in the USA because of factors other than the frequency of resistant varieties in the different areas.
- (4) Resistant varieties may be attacked by pest populations to which they never were resistant. For example, some populations of potato cyst nematodes have been found to attack eelworm-resistant potato varieties, and these were

at first considered to be true resistance-breaking biotypes (pathotypes) of *Globodera rostochiensis* (Howard, 1972). Some populations were later identified as *G. pallida*, a closely related species, to which the varieties had never been resistant; these populations are not, therefore, true resistance-breaking biotypes. Such populations of apparently resistance-breaking biotypes have been encountered with many different pests of several crop plants. Dr. V.E. Eastop (personal communication) considers that many reports of such 'false' resistance-breaking biotypes are attributable to misidentification of the pests concerned. It is very important, therefore, that pest populations that are able to attack previously resistant varieties are correctly identified, to determine whether or not they are true resistance-breaking biotypes. The strategy of subsequent selection and breeding operations will depend very much on the result of that identification. If a closely related pest species is responsible for the apparent 'breakdown' of resistance, the breeding programme must be extended accordingly to include screening for resistance to that species.

One of the main reasons for the relatively few cases of true resistance-breaking variants of animal pests may be that pests usually have fewer propagules than plant pathogens, in which genetic variation can be expressed. A host plant may support millions of pathogenic bacterial cells, or sufficient mycelium of a fungal pathogen to produce hundreds of thousands of spores. In contrast, the numbers of individual pest animals, even insects and nematodes, per plant can generally be measured in hundreds rather than thousands. This very great discrepancy between pathogens and animal pests, in the number of individual propagules per plant, is probably one of the major factors contributing to the relative unimportance of resistance-breaking biotypes of animal pests.

Another factor is that resistance of crop plants to pests is usually not responsive and is attributable to resistance mechanisms which existed in the host plant before the pest came into contact with it. There is usually no 'lock and key' interaction, dependent on a simply inherited resistance mechanism, between most pests and their host plants. This situation contrasts with the hypersensitivity type of responsive resistance mechanisms which are frequently encountered in interactions between fungi, bacteria and viruses and their host plants. An animal pest is, therefore, unable to circumvent most resistance mechanisms easily, and many types of pest resistance are, therefore, equivalent to non-race-specific types of resistance to plant pathogens.

Types of Resistance to Pests

Although vertebrate pests cause enormous damage to crop plants, there has been little progress in breeding varieties that are resistant to them or in characterizing types of resistance. This Section will, therefore, describe only those types of resistance to arthropod and nematode pests that have received considerable attention from the plant breeder.

Resistance to animal pests can be caused by one or more of several different kinds of resistance mechanisms. Painter (1951) recognized three main kinds of resistance to insect pests, that can be expressed by host plants. These are: (1) **non-preference**, which is shown by plants that are unattractive or unsuitable for colonization or oviposition by an insect; (2) **antibiosis**, which adversely affects the development or reproduction of insects; (3) **tolerance**, which enables

Table 10.1 A CLASSIFICATION OF DIFFERENT TYPES OF RESISTANCE TO ANIMAL PESTS

<i>Type of resistance</i>	<i>Characteristics</i>
Pest avoidance	The host plant tends to escape infestation
Non-preference (non-acceptance)	The pest tends not to stay to feed or reproduce on the host plants
Antibiosis	The growth rate or fecundity of the pest is adversely affected
Tolerance	The host plant is less damaged

a host plant to withstand an attack by insects without suffering severe damage. A fourth type of resistance, **pest avoidance**, which is expressed as a tendency to escape infestation, should also be considered. The main characteristics of each of these types of resistance, which apply equally to all groups of animal pests, are summarised in *Table 10.1*, and are described in detail below.

PEST AVOIDANCE

Some plants avoid infestations because they are not at a very susceptible stage when pest populations are at their peak. For example, some apple varieties do not become infested by several species of insect pests because their buds do not break until after the main hatching or emergence period of the pest (Briggs and Alston, 1969). This kind of resistance can greatly reduce the amount of pest damage that occurs.

NON-PREFERENCE

Any inherited feature of a host plant which discourages the feeding, colonization or oviposition of an animal pest makes it 'non-preferred' by that pest. The term 'non-acceptance' has been suggested by Van Marrewijk and De Ponti (1975) to describe this type of resistance. Non-acceptance is undoubtedly a more accurate term because, in most of the known examples of this type of resistance, insects will not 'accept' a resistant host plant even if there is no alternative source of food. It is not, therefore, usually a question of a pest preferring one host plant to another but rather that resistant plants are less acceptable than susceptible plants as hosts. However, Painter's nomenclature of types of resistance has been generally accepted (Pathak and Saxena, 1976) and the term 'non-preference' will therefore be used in this book in the sense of 'unacceptability'.

Non-preference to a pest can be expressed in many different ways. For example, non-preference resistance of raspberries to the raspberry aphid (*Amphorophora idaei*) is so strong that aphids quickly walk off resistant plants, a clear case of non-acceptance (see page 366). In sugar beet, where the effects of non-preference resistance to aphids are less clear-cut than in raspberries,

aphids do not walk off resistant plants but they feed for shorter periods and are generally more restless than on susceptible plants (Russell, 1966a; Lowe and Russell, 1969). The result of this resistance is that aphids do not flourish or reproduce to the same extent on resistant plants. The term 'resistance to aphid settling' was used by Russell (1966b) to describe this particular kind of non-preference.

Non-preference may be attributable to morphological, physiological or biochemical factors in the host plant. The presence of hairs (pubescence) on the leaves is associated with resistance to many insects, for example in cereals to the cereal leaf beetle (*see* page 329), in cotton to jassids (*see* page 337), and in turnips to the turnip aphid (Barnes and Cuthbert, 1975). It is uncertain, however, whether or not there is a direct, causal relationship between leaf pubescence and resistance (Pathak and Saxena, 1976). Oviposition of *Chilo suppressalis*, the striped moth borer, is less on pubescent than on glabrous rice varieties, but artificial removal of hairs did not affect the resistance of these varieties (Pathak, 1977). Gibson (1971a, b; 1976a, b) found that aphids and Colorado beetle larvae become trapped in gummy exudates from hairs on the leaves of certain *Solanum* species, so that they are unable to feed or reproduce.

The general size, shape and colour of a plant can contribute to the degree of non-preference shown by a pest. Red cabbage and red-leaved Brussels sprouts are less favoured by the butterfly, *Pieris rapae*, and certain other *Lepidoptera* for oviposition than are green varieties (Dickson and Eckenrode, 1975; Dunn and Kempton, 1976). Insects are particularly sensitive to the green and yellow parts of the spectrum of visible light, and butterflies are presumably attracted more to green and yellow plants than to red plants. This preference could, perhaps, be exploited by plant breeders in developing non-preferred varieties.

Mechanical obstruction to feeding or oviposition, because of toughness or thickness of plant tissues, seems to be important in reducing insect infestations. For example, the thickness of the leaf and the toughness of the vascular tissues can affect the level of resistance to jassids in cotton (Batra and Gupta, 1970). Wheats with solid stems are resistant to the wheat stem sawfly, partly because the eggs of *Cephus cinctus* do not hatch on them as well as they do on other varieties (*see* page 328). Nevertheless, no definite causal relationships between non-preference resistance to pests and morphological features of plants have been established in these or in other, similar, instances.

Some biochemical plant compounds, such as some essential oils in mite-resistant tomatoes, act as repellents to pests (Cantello, Boswell and Argauer, 1974). Nitrate ions, in the form of ammonium nitrate, apparently deter the feeding of a weevil, *Sitona cylindricollis*, in sweet clover (Akeson, Haskins and Gorz, 1969). Saponins in the roots of some lucerne varieties act as a strong feeding deterrent for the grass grub, *Costelytra zealandica* (Sutherland, Hood and Hillier, 1975).

Insects respond to various feeding stimuli in selecting their host plants (Kennedy, 1965) and the absence of such stimuli would presumably contribute to non-preference types of pest resistance. Pathak and Saxena (1976) cite several examples of plant compounds, including sugars, amino acids and vitamins, which can act as feeding stimulants of pests. Resistance of a rice variety, Mudgo, to the brown planthopper (*Nilaparvata lugens*) has been attributed to a low concentration of asparagine, which may act as a feeding stimulant to this pest (*see* page 345). Host selection by the cabbage aphid, *Brevicoryne brassicae*,

is stimulated by the presence of a mustard oil glucoside, sinigrin, in the leaves; a low concentration of sinigrin is apparently associated with a high level of resistance to this aphid in brassicas (see page 370). Flying sweet-clover weevils (*Sitona cylindricollis*) are attracted by coumarin, which is a constituent of their main food plant, *Melilotus* spp. (Thorsteinson, 1960).

ANTIBIOSIS

The rate of population increase of a pest on a host plant is reduced by antibiosis because it causes the death of the pest, or decreases its rate of development or reproductive potential. Plants can express many kinds of antibiosis, some of which involve differences in morphology and others, chemical factors. Responsive resistance mechanisms, which are triggered off by contact between a host plant and a parasite, are rare in relationships between plants and animal pests, and most resistance factors already exist in the host plant before the onset of pest attack. This contrasts with the relationship between plants and many fungal, viral and bacterial pathogens, in which responsive resistance mechanisms, including hypersensitivity, can be very important.

It is sometimes very difficult to distinguish between non-preference and antibiosis. This is partly because the same resistance mechanisms often seem to affect both the acceptability of a host plant by a pest and its subsequent development on it. For example, the morphological features of the cotton plant which determine the thickness and toughness of the leaf tissues probably contribute to both non-preference and antibiosis. Another factor contributing to confusion between non-preference and antibiosis is that they can both cause pest population decreases. Thus, resistance to settling of *Myzus persicae* on sugar beet, which is a form of non-preference, makes aphids restless and they do not flourish and reproduce on resistant plants to the same extent as they do on susceptible plants (see page 338). It is particularly difficult to differentiate between the effects of a continuous expression of non-preference such as this, and those of antibiosis.

Resistance of wheat to the stem sawfly is apparently caused by antibiosis which is based on morphological features of the host plant. The growth and development of sawfly larvae is retarded in the solid stems of resistant varieties; susceptible varieties, on the other hand, have hollow stems (Holmes and Peterson, 1957; Wallace, McNeal and Berg, 1973). Development of a stem borer, *Chilo suppressalis*, is adversely affected in rice varieties with a high silica content because the mandibular mouthparts of the insects are worn away in trying to feed on silica-impregnated plant tissues (Djamin and Pathak, 1967).

Biochemical factors are usually much more important than differences in morphology in determining the level of antibiosis to an animal pest. Exudates from glandular leaf hairs of many plants in the Solanaceae, including several species of *Solanum* and certain tobacco varieties, are toxic to a wide range of insect and mite pests (Gibson, 1976b). Exudates from secretory trichomes on the leaves of *Medicago disciformis* inhibit the alfalfa weevil (*Hypera postica*). These antibiotic exudates kill the weevil at high concentrations and retard its development at lower concentrations (Shade, Thompson and Campbell, 1975). Beck (1965) found that three chemicals are associated with the resistance of maize to the European corn borer, and it has been suggested that varietal

differences in the concentration of at least one of these might be used as a selection criterion in breeding for resistance to *Ostrinia nubilalis* (see page 347). Resistance to many insect pests of cotton is associated with high concentrations of a polyphenolic compound, gossypol, in the plant tissues (see page 335). High concentrations of benzyl alcohol are associated with resistance to greenbugs in wheat and barley (see page 327), and aphid-resistant varieties of alfalfa contain high concentrations of saponins in the leaves and stems. Leaves of *Lycopersicon hirsutum* f. *glabratum*, which is resistant to several pests including the tomato fruitworm, carmine red spider and tobacco flea beetle, contain a highly active, ethanol-soluble antibiotic compound that is lethal to these pests when fed to them in artificial diets (Fery and Cuthbert, 1975).

Much of the evidence in favour of a causal relationship between specific chemicals in a host plant and resistance to particular pests is circumstantial. Nevertheless, the indications are often sufficiently strong to justify a greater effort to obtain more evidence of such relationships, so that chemical assays could be used in selection for resistance. It might also be possible to develop new pesticides based on some of the natural compounds responsible for host-plant resistance.

Most of the antibiotic compounds that are known to occur in host plants retard the growth and development of pests. In some cases, however, such compounds have more specific effects as, for example, those involved in the resistance of potatoes and barley to cyst nematodes. Eggs of both these pests hatch near the roots, and larvae invade the root tissues of resistant and susceptible plants alike. However, no mature females (cysts) are produced on resistant plants, either because the larvae die or because they are predominantly male. Resistant plants can, therefore, be attacked by nematodes but no egg-containing cysts are produced on them and the nematode populations in the soil subsequently decline. The nature of the resistance mechanisms involved is not understood, but the chemicals responsible for this antibiosis may inhibit the formation of giant cells which appear to be essential for the development of mature females.

TOLERANCE

Tolerance does not in any way restrict or hinder the colonization of a host plant by a pest nor does it affect the development or reproduction of that pest. It does, however, reduce the damage to the host plant that is caused by a pest. A tolerant variety will grow more normally and produce higher yields than a sensitive (intolerant) variety when they are both infested to the same extent by a pest.

Tolerance to pests has been demonstrated with aphids in sugar beet (see page 338) and in brassicas (see page 369), and with greenbugs in cereals (see page 327). The leaves of some breeding lines of sugar beet quickly showed irreversible wilting when they were infested with large numbers of *Myzus persicae*, whereas leaves of other lines remained turgid under the same conditions. Several varieties of Brussels sprouts suffer little damage even when they are heavily infested by *Brevicoryne brassicae* (the cabbage aphid) although others are severely affected by small aphid populations. The nature of tolerance to aphids in sugar beet and brassicas has not been established. Greenbugs debilitate cereal plants by sucking sap from them, but they can also damage host

plants by injecting a toxin into the leaves while feeding. Some cereal plants are less damaged by this toxin, presumably because they are less sensitive to its effects than other plants, or can metabolize it to non-toxic compounds.

Non-preference, antibiosis and tolerance to an animal pest are sometimes expressed simultaneously in a single host plant. For example, some varieties of Brussels sprouts express all three types of resistance to the cabbage aphid. Such combinations of resistance types can be very effective in controlling pest attack and reducing the damage which can be caused by it.

Sources of Resistance

The same general principles apply to breeding for resistance to pests as for breeding for resistance to fungal, bacterial and viral diseases. This applies equally to types and sources of resistance.

One of the most successful programmes of screening collections for resistance to an animal pest was carried out by Ellenby (1954) in a search for resistance to potato cyst nematodes in the Commonwealth Potato Collection. He found resistance to what is now known to be a pathotype of *Globodera rostochiensis* in several Andigena clones of *Solanum tuberosum* and in a wild diploid species, *Solanum vernei*. Following this discovery, many programmes of breeding resistant potato varieties were set in train in the UK and several other parts of the world. Howard, Cole and Fuller (1970) screened the Andigena part of the Commonwealth Potato Collection again in an attempt to find resistance to what is now known to be the other species of potato cyst nematode, *G. pallida*, to which the original selections were susceptible. They found that several accessions of Andigena potato varieties were resistant to *G. pallida*, and these were used as additional sources of resistance. Howard and his co-workers emphasized the advantages of using resistance from Andigena sources, because it is much easier to obtain resistant varieties with good commercial qualities from cultivated potatoes than from wild species such as *Solanum vernei*. This underlines an important general rule, that resistance should be sought within cultivated varieties rather than in wild species, from which many undesirable traits may be transferred with the resistance genes.

The resistance to pests of about 2000 apple varieties, which comprise the UK National Fruit Trials, was assessed by omitting the usual insecticide sprays in one year and estimating the amount of pest damage on each variety (Briggs and Alston, 1969). Fourteen apple varieties showed a very high level of resistance to the rosy apple aphid (*Sappaphis mali*) and three were immune to the green apple aphid (*Aphis pomi*). Many varieties were resistant to the apple sucker (*Psylla mali*) and to the apple sawfly (*Hoplocampa testudinea*). These examples show that sources of resistance to a particular pest may be readily available and accessible in world or national collections of germplasm.

Resistance can be more easily detected and exploited in breeding programmes with self-pollinating or clonally propagated crop species than with cross-pollinating crops; resistant plants will be true-breeding. In cross-pollinated crops there is usually considerable plant-to-plant variation in resistance because populations are genetically variable. When searching for resistance in these crops it is particularly important to ensure that infestation of the pest is uniform, so that plants which escape pest attack or damage by chance are not selected as sources

of resistance. Each plant in an outbreeding crop may be a distinct genotype and there is, therefore, no replication of a particular genotype. Selected plants which show resistance can be self-pollinated or crossed in pairs or small groups. The resulting progenies can then be screened for resistance and further selections can be made, to form the basis of a resistant variety. This was the method used initially in seeking and exploiting sources of resistance to aphids in sugar beet (Russell, 1966a).

Most of the sources of resistance to animal pests have been found within the crop species itself. Resistance derived from wild species, by interspecific crosses, has been exploited in only a few crops, such as potatoes, tobacco and raspberries. Resistance to both species of potato cyst nematodes has been derived from *Solanum vernei*, and resistance to the root-knot nematode in tobacco has been obtained from related wild species. Genes for resistance to the *Rubus* aphid in raspberry have been found in other species of *Rubus* and in cultivated raspberries (Keep, Knight and Parker, 1970). Sources of resistance to jassids have been detected in cultivated varieties of cotton, and also in related wild species of *Gossypium*; both sources of resistance have been exploited by plant breeders.

Inheritance of Resistance

It is not necessary for a plant breeder to understand the genetics of resistance to a pest before that resistance can be exploited in the development of resistant varieties. Indeed, many of the most successful programmes of breeding for resistance have been carried out without detailed information concerning the number of resistance genes involved, or knowledge about whether the resistance is expressed as a dominant or a recessive character. For instance, resistance to the striped stem borer of rice (*Chilo suppressalis*) is complex and is controlled by many genes, each of which has only a small effect (*see* page 344). There is little, if any, information about the genetic control of resistance to many other important animal pests, against which resistant varieties have been produced (*Table 10.2*).

Some kinds of resistance to pests are controlled by a single gene (monogenically), others by a few genes of major effect (oligogenically) and still others by many genes each of small effect (polygenically). Some of the resistance genes are dominant, others are recessive and their effects may be additive. A single gene codes for the production of a certain protein which fulfils a particular metabolic function in the cell, for example catalyzing a specific chemical reaction. Such a reaction may trigger off several other reactions, so that many different compounds are eventually produced as a result of the initial reaction. One or more of these compounds may be involved in resistance to a pest or pathogen, so that even monogenically controlled resistance may involve several distinct resistance mechanisms. A different major resistance gene would trigger off a different reaction, or chain of reactions, in which the same or different compounds would be formed. Thus, two non-allelic major genes for resistance to a particular pest or disease might control either two different resistance mechanisms or the same mechanism. Resistance-breaking biotypes of a pest circumvent or neutralize a **resistance mechanism** – they do not ‘overcome’ the resistance genes themselves, but the mechanisms that the genes control, directly or indirectly.

Table 10.2 GENETICS OF RESISTANCE TO PESTS OF CROP PLANTS

<i>Crop species</i>	<i>Pest</i>	<i>Genetic control of inheritance</i>	<i>Dominance of resistance</i>	<i>Known biotypes</i>
Wheat	Hessian fly	Oligogenic	5 dominant 5 recessive genes	Yes*
	Greenbugs	Monogenic	Recessive (+ modifiers)	Yes*
	Stem sawfly	Oligogenic	Dominant + recessive genes	No
	Cereal leaf beetle	Polygenic	Not known	No
Barley	Greenbugs	Oligogenic	2 dominant genes	Yes
	Cyst nematode	Monogenic	Dominant	Yes
Oats	Stem eelworm	Monogenic	Dominant	No
Cotton	Bollworms	Not known		No
	Boll weevils	Not known		No
	Jassids	Monogenic	Dominant (+ modifiers)	No
Alfalfa	Spotted aphid	Polygenic	Not known	Yes
	Pea aphid	Oligogenic	1 dominant, 1 recessive	Yes
Rice	Stem borers	Polygenic	Not known	No
	Planthopper	Monogenic	Dominant + recessive genes	Yes*
	Leafhopper	Monogenic	Dominant	Yes*
Maize	European corn borer	Monogenic or oligogenic or cytoplasmic	1 or more dominant genes	No
	Stem borers	Not known	Not known	No
	Corn earworm	Probably polygenic	Not known	No
	Leaf aphid	Polygenic	Dominant, additive	?
Potato	Cyst nematodes	Monogenic and polygenic	Dominant, additive (?)	Yes*
Clover	Stem eelworm	Polygenic	Not known	No
Tobacco	Root-knot nematode	Monogenic	Dominant	?
Rubus (raspberry)	Rubus aphid	Monogenic	Dominant	Yes*
Brassica	Cabbage aphid	Polygenic(?)	Not known	Yes
Lettuce	Root aphid	Cytoplasmic	—	No
Apple	Woolly aphis	Monogenic	Dominant	Yes*

*Includes 'resistance-breaking' biotypes adapted to particular crop varieties.

The number and type of mechanisms involved in resistance to a pest affect the stability or durability of resistance and are therefore much more important than the number of genes which control these mechanisms. In general, oligogenic

and polygenic types of resistance are likely to involve more resistance mechanisms than monogenic resistance and will, therefore, probably be more stable or durable. It is, presumably, more difficult for a pest to overcome several independent resistance mechanisms than one mechanism, and the presence of several mechanisms in a variety may thus delay or stop the development of resistance-breaking biotypes.

There are, therefore, many advantages in using complex types of resistance that involve several different resistance mechanisms, and which will, almost inevitably, be controlled by more than one gene. On the other hand, complex resistance has many counterbalancing disadvantages. Monogenic resistance to a pest, particularly if it is inherited as a dominant character, is simple to use in a breeding programme; differences between resistant and susceptible plants are usually clear-cut, and this greatly facilitates selection for resistance. Resistant and susceptible plants segregate at predictable ratios and the resistance gene concerned can be transferred easily from one host genotype to another. Information concerning the genetics of resistance to several important insect, mite and nematode pests is summarized in *Table 10.2*. Most types of resistance that have been used are controlled by one or a few genes. This is presumably because the breeders have concentrated their efforts on types of resistance where there are clear-cut phenotypic differences, and which can easily be handled in a breeding programme.

Although biotypes of many of the pests listed in *Table 10.2* are known to exist, very few of them are adapted to specific varieties of host plants. The other biotypes differ from one another, either in preferring particular species of food plants, in some morphological feature, or in degree of general vigour or size and are not, therefore, true resistance-breaking biotypes. Those types of resistance which have been 'overcome' by resistance-breaking pest biotypes have, almost invariably, been those which are controlled by major genes. Similar conclusions have been drawn from the occurrence of resistance-breaking variants of fungal, viral and bacterial plant pathogens.

However, some types of monogenic resistance to crop pests have not been overcome by resistance-breaking biotypes, even when resistant varieties have been exposed extensively to pest attack for many years. For example, jassid-resistant cottons and leaf beetle-resistant cereals have been widely grown for many years and these resistances must be classified as durable, although they are controlled by major genes. It is not understood why problems with resistance-breaking pest biotypes have been encountered with some monogenic resistances but not with others; however, three factors may be of particular importance. First, some varieties with monogenic resistance have not been widely grown or exposed to pest attack sufficiently for resistance-breaking biotypes to arise or be detected. Second, resistance-breaking biotypes of some pests may be less likely to occur or be disseminated than those of other pests. For example, resistance-breaking biotypes of stem eelworm (*Ditylenchus dipsaci*) have been identified, but have not become widespread or caused severe damage on resistant clover or oat varieties. Third, many types of resistance to pests are based on gross morphological features of the host plant, such as pubescence or solid stems, and these seem generally to be durable. Resistance-breaking biotypes have not been encountered with sawfly-resistant wheats with solid stems, with pubescent cereal varieties that are resistant to the cereal leaf beetle, or with

hairy jassid-resistant cotton varieties; all of these are examples of durable monogenic resistance.

In spite of the complexity of using polygenically controlled resistance to pests, *Table 10.2* lists several examples where such resistance has been used successfully. No resistance-breaking biotypes seem to have been able to adapt themselves to varieties with polygenically controlled resistance.

In order to achieve maximum durability, a plant breeder should try to combine several different types of resistance to a pest, for example non-preference, antibiosis and tolerance. He should also concentrate particularly on those types of resistance which are likely to be non-race-specific and therefore more durable. It should be realized, however, that increasing the complexity of breeding for resistance takes time and facilities which might otherwise be devoted to other important objectives, such as high yield and good quality. Unless damage by a particular pest is of overriding importance in a crop, it is unlikely that even a highly resistant variety will become popular with growers if it does not also have acceptable commercial and agronomic qualities. It is very important, therefore, that plant breeders should not neglect to select for these qualities while breeding for pest resistance.

Several characteristics of plants and animals are known to be controlled by genes in the cytoplasm, that is by extranuclear or extrachromosomal genes. Such influence of the cytoplasm on resistance to animal pests appears from the literature to be very rare, but not unknown. The presence of Texas cytoplasm can influence susceptibility of maize to *Ostrinia nubilalis*, the European corn borer (*see* page 349), and resistance to the lettuce root aphid is cytoplasmically controlled (*see* page 374). Tests for resistance to pests, using progenies of reciprocal crosses between resistant and susceptible plants, might reveal other examples of cytoplasmic influence on pest resistance.

Selection and Breeding Methods

The most appropriate testing and selection procedures to be used in breeding for resistance to a particular pest depend on several factors, including the breeding system of the host plant, the type and genetics of the resistance involved and the biology of the pest concerned.

In cross-pollinated crops (*Table 2.1*), such as maize, sugar beet and brassicas, individual plants in a population usually have different genotypes and there is considerable plant-to-plant variation in resistance to a pest or disease. It is, therefore, often desirable to select for improved resistance on an individual plant basis in these crops, and it is common practice to self-pollinate selected plants, or to cross them in pairs or small groups. The progenies of selections are then tested for resistance and further selections are made. These re-selections can be interpollinated in larger groups to produce 'breeding lines', or crossed with progenies of unselected plants to maintain a high level of genetic variability. Alternatively, selected plants can be self-pollinated in successive generations of re-selection so that resistant inbred lines are produced. Unfortunately, in many cross-pollinated crops, such inbreeding is followed by a very marked decrease in general vigour and productivity, which is often referred to as 'inbreeding depression'. Although the vigour of the parent material can often be regained,

or even exceeded, by crossing appropriate inbred lines, the effects of inbreeding can give misleading results in selection tests for resistance to pests and diseases. For example, inbred plants are often less attacked and damaged by pests than other plants, solely because they are small and do not grow well. For this, and several other practical reasons, it is often desirable to avoid close inbreeding; recurrent selection for resistance, in populations derived by interpollinating selected plants, is widely practised in outbreeding crops. This procedure can improve the level of resistance in the populations while maintaining an acceptable level of heterozygosity.

In self-pollinating crops, the progeny of a plant that has been selected for resistance to a pest, consists of genetically identical individuals, but segregation for resistance will occur in later generations. Resistant true-breeding lines can be selected for pest resistance in these later generations, using the pedigree or the bulk method (*Figure 2.1*). After several generations of selfing, each plant will be homozygous and no further selection for resistance will then be possible. This situation contrasts with that in cross-pollinated crops, where recurrent selection for resistance is possible because homozygosity is never achieved.

In self-pollinated crops, therefore, screening for resistance to a pest usually involves testing a number of individual, genetically stable genotypes, and retaining the more promising genotypes for retesting and multiplication. The level of resistance can be further improved only by crossing some of these genotypes and seeking genotypes that express greater resistance among the F_2 and later generations. In cross-pollinated crops, however, populations of genetically distinct plants are screened for resistance to a pest, and the most resistant plants are selected and usually interpollinated. The progenies of selected plants are then tested for resistance, and re-selections are made. In this way, populations of plants with a higher level of pest resistance are developed by recurrent selection.

Testing for resistance to pests can be carried out in the field or in the glasshouse; both types of test have been employed in many breeding programmes. When field tests are conducted under natural, uncontrolled environmental conditions, experimental errors can be so high that spurious differences in pest resistance between genotypes are obtained, or real differences in resistance are obscured. Nevertheless, field experiments have many advantages because they are not carried out under artificial conditions; in addition, larger numbers of host genotypes or breeding lines can usually be tested in the field than in the glasshouse, with the same amount of labour. On the other hand, experimental procedures in the glasshouse or laboratory can be standardized and controlled, so that reproducible results can often be obtained quickly and easily.

Natural epidemics of a pest can be exploited in breeding for resistance by laying down field trials in areas where pest attacks are most likely to occur. In the case of soil pests, experimental plant material should be planted in soil that is known to contain the pest concerned. This method has been used, for example, in screening oats and clover for resistance to the stem eelworm (*Ditylenchus dipsaci*). Large populations of soil pests can usually be maintained for testing purposes in particular fields by continuously cropping them with susceptible host plants.

Natural field epidemics of air-dispersed pests occur more frequently in some localities than others, either because they are near the breeding grounds or major overwintering sites of the pests, or because the prevailing environmental conditions are particularly suitable for the multiplication and dispersal of the

pests. Field tests for resistance of rice to lepidopterous stem borer larvae have been conducted out of season when there were few alternative oviposition sites for adult female moths. This resulted in heavy natural infestations of stem borers on the test plants and greatly facilitated selection for improved resistance (see page 342).

In glasshouse tests, and in many field experiments, it is not possible to rely on the natural occurrence and spread of pests, and infestations have to be artificially induced. This can be achieved in two main ways: first, by introducing pests into the glasshouse or field trial area, on the assumption that they will flourish and spread uniformly throughout the plant material to be tested; second, by infesting each plot or test plant with an approximately equal number of pest individuals. Many different inoculation methods can be used to screen breeding material for resistance to pests. For example, in testing for resistance to cyst nematodes in potatoes, tubers have been grown in small pots containing nematode-infested soil, or sterilized soil to which cysts were added (Howard, Cole and Fuller, 1970). Root balls were examined at about 14-day intervals and the number of nematode cysts on the roots of each plant was estimated. Resistant plants had no cysts and susceptible plants had usually more than 200 cysts per root ball.

In testing sugar beet lines for resistance to aphids in the field, a piece of Chinese cabbage leaf supporting about five *Myzus persicae* was placed on each plant to be tested. When this leaf piece wilted, the aphids walked off it, on to the test plants. In other experiments, pieces of infested Chinese cabbage leaves were placed between rows of test plants, because this simulates natural infestation more closely than the previous method, and is less laborious (Lowe, 1973). In glasshouse tests, individual sugar beet plants were infested with five apterous aphids, either by transferring them directly to the test plants from infested plants by means of a soft paintbrush, or after temporary storage in gelatine capsules (Lowe, 1974a). Infested test plants were then covered with a transparent plastic beaker for several hours to confine the aphids to the vicinity of the test plant. This was necessary because *M. persicae* often shows a tendency to walk off sugar beet plants to which they have been transferred, and this is not necessarily related to the resistance of the plants concerned.

Initial screening of alfalfa for resistance to the spotted alfalfa aphid was carried out in seedboxes in the glasshouse (see page 354). Seedlings were heavily infested with aphids and aphid damage was assessed about four weeks later; susceptible plants were usually killed after this period, whereas resistant plants were still growing vigorously. Resistant seedlings were transplanted into pots and re-infested after a period of recovery, when further selections were made. Cages made of transparent plastic were also used to contain aphids on individual leaves.

Screening for resistance to pests in the field can often be facilitated by inter-planting rows of susceptible varieties between rows of test plant material. This method has been used in breeding for resistance to stem borers in rice. In other tests, rice plants were infested with egg masses of *Chilo suppressalis*, each containing about 60 eggs, and the number of eggs that hatched on each plant was recorded (Pathak *et al.*, 1971). In glasshouse tests, larvae were transferred from egg masses to test plants by means of a soft brush soon after they had hatched; the extent of damage on each test plant was then recorded and compared with the amount of damage on susceptible control varieties.

It can be seen from this brief account of testing procedures that many dependable methods have been devised for screening for resistance to invertebrate pests in the field and glasshouse. It should be realized that, with diseases as with pests, glasshouse and field tests are complementary to each other, and that there are advantages and disadvantages in both testing systems. It is important that field tests, using natural pest infestations, should be carried out wherever possible, to check the results of artificially inoculated field or glasshouse selection procedures.

Control of Animal Pests by Resistant Varieties

Attempts to breed pest-resistant varieties have mainly been concentrated on nematode and arthropod (particularly insect) pests. There have been few concerted attempts to find sources of resistance to vertebrate pests (birds and small mammals) which are probably the most damaging of all. An exception is the attempt by the United States Fish and Wildlife Service to discover lines of maize resistant to 'blackbirds', which cause serious losses of grain in several States. Inherited differences between lines of maize in palatability to 'blackbirds' have already been found (see page 353). This is not entirely surprising because, almost without exception, sources of resistance to nematodes, mites and insects have been found whenever they have been sought. Recent work by Dr R.E. Chapman (personal communication) on host plant selection by grasshoppers suggests that it may be possible to breed for resistance to solitary grasshoppers and even to locusts in a number of crops. The possibilities of developing resistant varieties to any crop pest should be examined, no matter how unlikely those possibilities may seem.

THE EFFECTIVENESS OF RESISTANCE

Effective resistance to cyst, stem and root-knot nematodes, has been found and exploited in several crop species. For example, varieties of potatoes and barley resistant to cyst nematodes of the genera *Globodera* and *Heterodera* are extensively grown and are beginning to play an important part in the control of these pests. Resistance to *H. avenae* in barley and to *G. rostochiensis* and *G. pallida* in potatoes is based on the inhibition of development of mature females on resistant plants and, in consequence, the nematode population in the soil is reduced after resistant varieties have been grown. Both these resistances are controlled by dominant major genes and are therefore easy to manage in breeding programmes (see pages 332 and 357). Several biotypes (pathotypes) of these nematode species can complete their life cycle on host genotypes with particular resistance genes. Nevertheless, resistant varieties have given a satisfactory control of cyst nematodes in several European countries, particularly when they have been used in conjunction with other control measures such as appropriate crop rotations. Resistant varieties have so far been used only for a short time and there are bound to be occasional localized failures of resistant varieties to control cyst nematodes, because of the occurrence of resistance-breaking pest pathotypes. However, rapid and widespread breakdowns of control

are unlikely because new biotypes of soil-borne pests are usually disseminated much more slowly than are those of air-borne pests.

Attempts to breed for resistance to cyst nematodes have been much less successful in sugar beet than in potatoes and barley. Although resistance of the type used successfully in cereals and potatoes has been identified in wild relatives of the sugar beet, it has proved very difficult to transfer this resistance from the wild parent without also transferring many undesirable characteristics. Other kinds of partial resistance to *Heterodera schachtii* have been found but nematode-resistant varieties with acceptable yields and quality have not yet been produced. It is unlikely that resistant sugar beet varieties will play a significant part in controlling cyst nematodes in the foreseeable future.

Several varieties of oats and clover, showing partial resistance to *Ditylenchus dipsaci* (stem nematode), have helped to decrease damage by this pest in many parts of the world (see pages 333 and 362). Tobacco varieties resistant to the root-knot nematode (*Meloidogyne* spp.) have also been extensively used to control this very important pest (see page 364).

The scale and scope of work on breeding for resistance to insect pests and on studying the nature of resistance has greatly increased since the mid-1950s. This increased interest in insect resistance was stimulated largely by the successes of the late Dr. R.H. Painter and his team of plant breeders and entomologists in Kansas. Much excellent work on insect resistance has been carried out since then but much of it is too recent to have had a significant impact on agriculture or horticulture. This is because there has either been insufficient time to produce commercially acceptable resistant varieties or such varieties have not been grown extensively. However, most of this work should shortly come to fruition, and insect-resistant varieties may then become even more important in crop protection, particularly because of the expense and potential loss of effectiveness of many of the other control measures. Pesticide-resistant variants of many important crop pests are becoming increasingly common and more widely distributed, and resistant varieties are urgently needed to underpin chemical control measures.

It is important to remember that there can be quite different reasons for attempting to breed for resistance to a particular crop pest. Three important objectives are, first, to reduce direct feeding damage by the pest, second, to decrease the spread of pest-transmitted viruses or mycoplasmas and third, to reduce pest infestation for cosmetic reasons. With the first two objectives it is usually not necessary to have complete resistance, because a few pests and some damage can be tolerated in most crops. Partial resistance can be considerably more useful in the field than it might seem from the results of laboratory and glasshouse tests, because it can greatly increase the effectiveness of chemical and biological control measures. Where the main objective of breeding for pest resistance is to decrease spread of viruses, certain types of resistance may be more desirable than others. For example, a 'walk-off' non-preference type of resistance to an insect vector might theoretically increase the spread of non-persistent viruses within a crop. Vectors might acquire such a virus from an infected insect-resistant host plant during short feeds, and quickly walk off in search of a more acceptable plant; in so doing they could transmit the virus to adjacent plants during short feeds. However, the author knows of no case where virus spread has actually increased as a result of insect resistance. Indeed, a 'walk-off' type of resistance to the raspberry aphid (*Amphorophora idaei*) has

been shown to give a very effective control both of persistent and non-persistent viruses of raspberries in the field (Jones, 1976).

In developed countries, no trace of insects or insect damage is tolerated by the consumer in some vegetables and fruits. Partial resistance would, clearly, give an insufficiently good control for such 'cosmetic' reasons, unless other control measures were also used. Complete resistance to many insect pests has been achieved in several vegetable and fruit crops, for example to the raspberry aphid in raspberries (see page 365) and to the root aphid of lettuce (Dunn and Kempton, 1974).

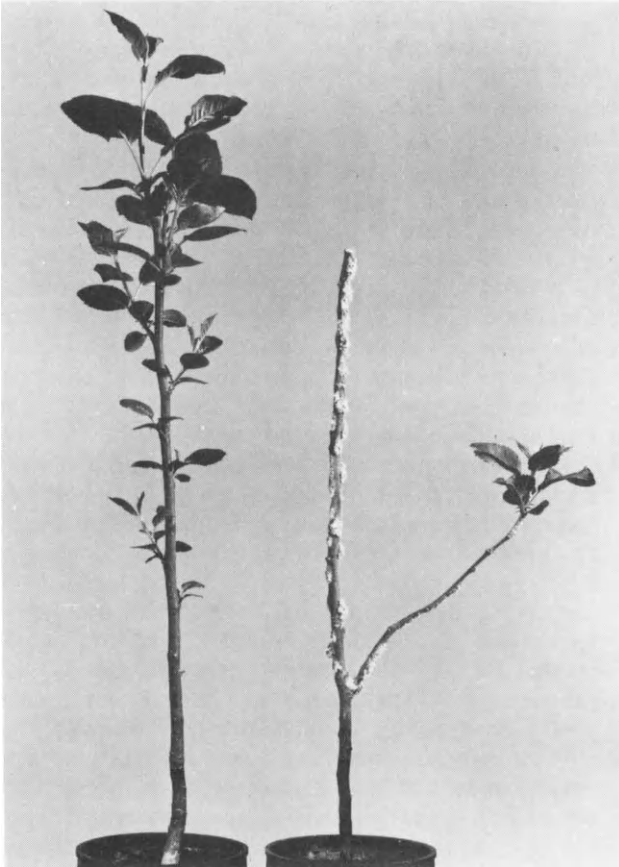


Figure 10.2 Apple seedlings repeatedly inoculated with nymphs of the woolly aphid Eriosoma lanigerum. A resistant seedling (left) carrying the genes Erer is infested with only five aphids whereas a susceptible seedling (genes erer) has more than 1000 aphids. (By courtesy of East Malling Research Station)

Many invertebrate pests have been very effectively controlled by resistant varieties in a wide range of crop species for many years. For example, *Phylloxera virifolia*, a Hemipteran pest of grapevine roots, threatened the existence of the entire French grape and wine industry in the 1860s. The industry was saved

by a programme of grafting susceptible vines on to *Phylloxera*-resistant rootstocks, and this resistance has given an excellent control of the pest ever since. Resistance to the woolly apple aphid (*Eriosoma lanigerum*), which was derived from the variety Winter Majestic has effectively controlled this pest for more than 100 years. Although resistance-breaking biotypes of the woolly aphid have recently been reported in Australia and South Africa (Sen Gupta and Miles, 1975) the effectiveness of control has not yet been significantly decreased (Figure 10.2).

DURABILITY OF RESISTANCE

The threat of resistance-breaking biotypes of animal pests must always be considered. Before the importance of biotypes can be assessed in relation to the performance of pests in resistant varieties in the field, it is necessary to differentiate between the four main kinds of biotypes which may be involved (see page 305).

The longevity or durability of pest resistance shown by a variety will depend largely on whether true resistance-breaking biotypes appear in the pest populations, and how quickly they build up and are disseminated. Fortunately, resistance-breaking biotypes of most pests have not been as much of a problem to the plant breeder in practice as have physiologic races of fungal and bacterial plant pathogens. Even where such biotypes have been recognized, they have not so far led to the withdrawal of resistant varieties from commerce, as has often been the case with physiologic races of cereal rusts and mildews, for instance. There are many possible explanations for this difference in the durability of resistance to animal pests and pathogens. Animal pests may be less variable genetically than fungal or bacterial plant pathogens, although there is no direct evidence that this is the case. However, as has already been stated, the average number of animal pests which infest a host plant is usually much less than the number of fungal or bacterial propagules on an infected host; this situation must lead to a greater probability of resistance-breaking variants in pathogens than in animal pests, and to an increased likelihood that such variants with the necessary fitness to survive will soon develop. This fitness can be very important because 'unfit' variants will quickly disappear from a pest population in the absence of host plants on which they have a selective advantage.

True resistance-breaking biotypes seem to be more common in nematodes and Hemiptera, particularly aphids, than in moths and butterflies (Lepidoptera), beetles and weevils (Coleoptera), or sawflies (Hymenoptera). This may reflect the very close and specific associations between nematodes and Hemiptera and their host plants.

Certain types of resistance have given rise to resistance-breaking biotype problems more frequently than others. Resistance to most animal pests does not, apparently, involve responsive resistance mechanisms of the host plant, the mechanisms existing in the host before the start of an attack by a pest. There is usually no specific interaction between a host and a pest as there is with many plant pathogens. Many resistances to fungal and bacterial pathogens involve an active response of the host plant to infection, such as hypersensitivity.

Resistance that is associated with particular morphological or anatomical features of the host plant has usually been more stable or durable than many other kinds of resistance. For example, resistance to several insect pests is associated with an abundance of hairs on the aerial parts of the host plant (Table 10.3). This pubescence is known to deter oviposition by some insect

Table 10.3 THE RACE SPECIFICITY OF SOME MECHANISMS OF RESISTANCE TO VARIOUS INSECT PESTS

<i>Resistance mechanisms</i>	<i>Pest</i>	<i>Resistance-breaking biotypes</i>
Leaf pubescence	Cereal leaf beetle	—
	Jassids	—
	Stem borers	—
Solid stems	Wheat stem sawfly	—
High silica content	Rice stem borers	—
Nutritional deficiencies	Rice planthoppers	+
Chemical antibiosis factors		
gossypol	Cotton pests	—
saponins	Alfalfa aphids	—
benzyl alcohol	Cereal greenbugs	+
DIMBOA	European corn borer	—
Unknown factors	Hessian fly	+
	Raspberry aphid	+
	Brassica aphid	+

pests on the leaves and stems, and can also retard the development of insect larvae on the surface of plants by impeding their movement. The genes that control the degree of pubescence may also be closely linked with genes that control characteristics of the host plant, which affect in other ways the development of the pest concerned.

Some chemical antibiosis mechanisms have also been associated with durable resistance to pests. Cotton plants containing high concentrations of a poisonous polyphenolic pigment, gossypol, are resistant to several insect pests, although a direct causal relationship has not been established. Maize varieties that are resistant to *Ostrinia nubilalis*, the European corn borer, contain high concentrations of 2,4-dihydroxy-7-methoxy-2H-1,4-benzoxazin-3(4H)-one (DIMBOA), which is highly distasteful to this pest. Although the presence of antibiotic compounds such as gossypol or DIMBOA can be a useful indication of resistance to some pests, they are unlikely to be the sole causes of resistance. Nevertheless, the presence of gossypol, which appears to be a broad-spectrum poison, seems to deter feeding of several important cotton pests and may be largely responsible for the durability of pest resistance in cotton.

Resistance-breaking insect biotypes have been encountered more frequently with resistance controlled by a single gene than with polygenically controlled resistance (Table 10.4). Nevertheless, some monogenically controlled resistances to pests have been very durable, for example to stem eelworm in Grey Winter

oats (see page 333). On the other hand, resistance-breaking biotypes of *Brevicoryne brassicae*, the cabbage aphid, have been able to attack rape varieties with polygenically controlled resistance to aphids in New Zealand and England (Dunn and Kempton, 1972).

Although true resistance-breaking biotypes of many invertebrate pests have been reported, resistant varieties have nevertheless usually given a good control of most pests in the field. Reports of the occurrence of biotypes should not, therefore, discourage plant breeders from trying to develop resistant varieties.

Table 10.4 THE GENETICS OF RESISTANCE TO SOME INSECT PESTS IN RELATION TO RACE SPECIFICITY

<i>Genetics of resistance</i>	<i>Pest</i>	<i>Crop</i>	<i>Resistance-breaking biotypes</i>
Monogenic	Greenbugs	Wheat	+
	Aphids	Raspberry	+
	Planthoppers	Rice	+
Oligogenic	Greenbugs	Barley	+
	Jassids	Cotton	—
	Pea aphid	Alfalfa	—
	Stem sawfly	Wheat	—
	Hessian fly	Wheat	+
	Corn borer	Maize	—
Polygenic	Leaf beetle	Cereals	—
	Stem borers	Rice	—
	Leaf aphid	Maize	?
	Earworm	Maize	—
	Spotted aphid	Alfalfa	—
	Aphids	Brassicas	+?

From past experience it seems that such biotypes will not significantly decrease the potential usefulness of resistance in the field for many years, and it is very unlikely that they will cause a complete breakdown in resistance over a wide area.

General Conclusions

Many varieties with good resistance to an economically important pest have not been fully exploited because they yield less, or are of poorer quality, than many susceptible varieties. This emphasizes a general principle, the importance of which is not always appreciated by zoologists, particularly those who are concerned with the more theoretical aspects of relationships between a pest and its host plant; pest resistance is just one of many breeding objectives, and a variety with excellent resistance to pests and diseases will not become generally accepted unless it yields well and is of good quality in the absence of pest attack. The nature and genetics of resistance should, therefore, not be so complex that the plant breeder cannot easily manage to combine resistance with other desirable characteristics in a breeding programme.

In summary, resistant varieties of many crop species have given a very effective, although usually only partial, control of pests and often also of viruses that can be transmitted by these pests (Painter, 1968; Pathak, 1970). There is obviously a very great potential for plant breeders and entomologists to develop pest-resistant varieties. Although it can be very helpful to understand the mechanisms responsible for resistance to pests, this information is by no means essential for the development of successful pest-resistant varieties. This is shown clearly by the many past successes of breeding for resistance, using empirical methods of selection and breeding.

A full-scale breeding programme with the objective of developing highly resistant or immune varieties can be justified only in the case of the most damaging pest species. For the rest, it is probably sufficient to avoid the production of varieties that are very susceptible to any pest or disease which is likely to threaten the crop species concerned. Such breeding programmes, which involve discarding the most susceptible breeding material rather than selecting the most resistant, can help to produce commercially acceptable varieties which are unlikely to be severely damaged by pests and diseases. In this way, plant breeders can often make a major contribution to crop protection, without the need to neglect other breeding objectives while trying to achieve very high levels of pest and disease resistance.

Very considerable resources and effort are now being employed in most parts of the world in studying the relationships between pests and their host plants, and in developing pest-resistant varieties. The availability of such varieties will make a very significant contribution to the control of pests in all major agricultural and horticultural crops, and should be the basis of all integrated control programmes.

References

The references cited in this Chapter, together with those for Chapter 11, are listed in References – Part IV, pages 379–400.

EXAMPLES OF RESISTANCE TO ANIMAL PESTS

Wheat, Barley and Oats

Small-grain cereals are susceptible to a large number of vertebrate and invertebrate pests, and resistant varieties are often the main method of controlling some of the latter. Progress in breeding for resistance will be illustrated by reference to the following six different types of invertebrate pests: Hessian fly (wheat), greenbugs (wheat and barley), stem sawfly (wheat), cereal leaf beetle (wheat), cereal root nematode (wheat and barley) and stem eelworm (oats).

HESSIAN FLY

The Hessian fly (*Mayetiola destructor*) was introduced into the USA during the latter half of the eighteenth century, and quickly became an important pest in the major wheat-growing regions although, in its native Europe, Hessian fly does not seem to have been very destructive. Adult flies emerge from pupae in the soil and lay very large numbers of eggs on the upper surface of young wheat leaves. The larvae which hatch from the eggs attack wheat plants, and in severe infestations can kill them or make them more prone to frost damage; more often the straw is damaged so that lodging occurs. Heavy attacks by Hessian fly can, therefore, seriously decrease the yield and quality of grain (Painter, 1960).

A search for resistance was initiated in Kansas in 1914, and several resistant varieties have been developed. Kawvale, a variety derived from a soft-wheat variety Indiana Swamp, was released as a Hessian fly-resistant variety in 1928. It was superseded by Pawnee, a higher quality variety with improved resistance, which was derived from a cross between Kawvale and a susceptible variety Tenmarq. Pawnee gave good yields of grain, even when heavily infested with Hessian fly. A variety with a greater level of resistance, Ponca, was released in 1951; in 27 field tests in Kansas only two per cent of Ponca plants were infested, compared with 48 per cent for Pawnee and 75 per cent for the susceptible variety Tenmarq. The growing of resistant varieties in Kansas since 1965 has resulted in the virtual disappearance of Hessian fly from the State, except in areas where susceptible varieties are still grown (Painter, 1966). In 1969, 24 different

resistant varieties were being grown on 3.5 million hectares in the USA (Gallun and Hatchett, 1969; Gallun, Starks and Guthrie, 1975); by 1974 the area of resistant varieties in the USA had increased to more than 6 million hectares (Gallun, 1977). New sources of resistance to Hessian fly have been discovered in varieties from Spain and Portugal and these are being used extensively in breeding programmes.

There are few published reports concerning the nature of resistance to this insect. Resistant wheats have unusually large silica deposits on the leaf sheaths (Miller *et al.*, 1960), but no causal relationships between silica content and resistance have been established. The genetics of this resistance has been more intensively studied than the resistance mechanisms. Resistance is controlled by a series of dominant or partially dominant genes (designated H_1 – H_8) and several recessive genes (Allen *et al.*, 1959; Abdel-Malik, Heyne and Painter, 1966; Patterson and Gallun, 1973). F_1 hybrids between resistant plants carrying the H_3 gene (which governs resistance to race C of *M. destructor*) and susceptible varieties, express a level of resistance intermediate to that of the parents. The resistance is expressed as reduced damage to the host plant (tolerance) and as antibiosis, the larvae being fewer and smaller on resistant plants.

There is a gene-for-gene relationship between the resistance genes of wheat and the virulence genes of the pest. Wheat varieties carrying the H_1 and H_2 genes are very resistant to Hessian fly in California, less resistant in Kansas and susceptible elsewhere in the USA; this is because biotypes capable of attacking varieties with resistance controlled by the H_1 and H_2 genes are absent from California. Eight biotypes, designated Great Plains and A to G, have been identified in the USA (Hatchett and Gallun, 1968; Hatchett, 1969; Gallun *et al.*, 1975). These biotypes vary in their ability to survive on resistant wheats. For example, the Great Plains biotype cannot survive on varieties carrying any of the eight resistance genes that have been identified so far. Biotypes E and F can attack wheats with resistance genes H_3 and H_4 , and H_5 and H_6 , respectively. Biotype D, on the other hand, is controlled only on wheat varieties carrying the H_5 gene (Gallun *et al.*, 1975). No biotype has yet been identified that can overcome the resistance controlled by all the H resistance genes.

Although it is likely that more biotypes of *M. destructor* will be discovered, resistance-breaking biotypes will not necessarily be a problem in the use of resistant varieties. Some races of the fly are less competitive than others in the field (Foster and Gallun, 1973) and populations of some resistance-breaking biotypes may therefore not reach damaging proportions. Nevertheless, some previously immune wheat varieties have been badly attacked by biotypes that were not previously common in parts of the USA, and the dangers of resistance-breaking biotypes should, therefore, not be underestimated (Gallun, 1977). The situation concerning biotypes of *M. destructor* is further complicated by the differential reaction of resistant wheat genotypes to specific biotypes at different temperatures (Sosa and Foster, 1976). Sources of resistance that will give an effective control of all known races over a wide range of different temperatures are now being sought. In the meantime, Gallun, (1977) advocates the use of single resistance genes in new varieties, and that these genes should be changed when resistant biotypes become common in the field.

The benefits that have already resulted from the growing of Hessian fly-resistant varieties have been very great. It has been estimated, for example, that the value of the wheat crop in the USA has been increased by many millions of

dollars annually through control by resistant varieties (Pathak, 1970; Gallun *et al.*, 1975).

GREENBUGS

Greenbugs, (*Schizaphis (Toxoptera) graminum*), are aphids that can cause widespread damage to wheat and barley in the central and south-western USA. This damage is apparently caused by toxins which are injected into the plant with the saliva as the aphids feed. In the Spring, heavy infestations of *S. graminum* can kill young wheat plants, but more usually, aphid feeding results in poor root growth, a reduction in tiller number and reduced grain yield and quality.

More than 7000 wheat genotypes have been tested for resistance by the US Department of Agriculture in Oklahoma and Kansas. Eight of these, all spring wheats, were selected as showing good resistance to greenbugs. Derivatives of *Triticum durum*, particularly Dickinson 485, which was selected from C13707, showed the highest expression of resistance. A significant level of greenbug resistance has been found in a Triticale (wheat $\times$ rye hybrid) line by Starks and Merkle (1977). Many barleys were highly resistant in these tests, including several from China, Korea and Japan, and the American variety Dicktoo.

Resistance to greenbugs in wheat is controlled by a single recessive gene, although modifying genes are also involved (Painter and Peters, 1956). Resistance in barley is controlled by a single dominant gene located on the centromere segment of chromosome 1 in the T1-6a translocation (Smith, Schlehuber and Curtis, 1962; Gardenshire, 1965; Gardenshire, Tuleen and Stewart, 1973). Both tolerance and antibiosis seem to be involved in the resistance of barley to greenbugs (Painter, 1967) and these types of resistance are probably independently inherited. On average, twice as many larvae are produced on susceptible as on resistant barley from the same number of adult aphids; this implies that populations would be three and 1500 times greater on susceptible plants than on resistant plants, after 15 and 50 days respectively, with the same initial population of adult aphids (Dahms, 1969). The effects of resistance might be even greater than this in the field, however, because the mortality of larvae, which is often a result of antibiosis, was not taken into account in these calculations (Pathak, 1970).

No correlation has been found between individual morphological characteristics of the host plant and resistance to greenbugs (Painter, 1967) and the mechanisms of resistance have not been identified. Resistant plants often contain higher concentrations of benzyl alcohol, which is toxic to greenbugs, than do susceptible plants (Juneja *et al.*, 1975; Juneja and Gholson, 1976). *S. graminum* is more restless on resistant wheat, barley and oat plants than on susceptible plants and this probably reduces feeding by the pest, thereby decreasing its fecundity and damage to the host plant (Starks and Burton, 1977).

Although selecting and testing for resistance can be carried out in the field, it is difficult to ensure that there will be enough greenbugs present in the plots to differentiate between host genotypes. For this reason, much of the work on breeding for resistance has been carried out in the glasshouse. Seedlings are each infested with a predetermined number of adult greenbugs, which are then allowed to feed and reproduce parthenogenetically. The number of aphids on each plant is recorded at regular intervals, together with the number of seedlings

killed by greenbugs. With barley, the greenbugs can be left on the surviving plants more or less indefinitely; the resistant plants will not be killed. With wheat, however, the insects must be controlled with insecticides as soon as the more susceptible plants have been killed, because even the most resistant seedlings will eventually succumb (Painter, 1967).

Considerable progress has been made in developing greenbug-resistant varieties in the USA, and several resistant barley varieties have been released. For example, Will was produced in Oklahoma by crossing the resistant Kearney with the susceptible Rogers. Painter (1967) predicted that widespread planting of the variety Will in Oklahoma and Kansas would reduce the greenbug population by about 50 per cent in resistant crops, and would help to protect adjacent crops of susceptible varieties by decreasing the general level of aphid populations within an area. Although biotypes differing in their ability to colonize wheat, barley and sorghum are known to occur (Wood, Chada and Saxena, 1969; Gallun *et al.*, 1975), resistance-breaking biotypes of *Shizaphis graminum* have not been a major problem in practice.

WHEAT STEM SAWFLY

The wheat stem sawfly, *Cephus cinctus*, was a serious pest of wheat in Canada and parts of the western USA until resistant varieties became widely grown. In badly infested crops, loss of grain yield has sometimes exceeded 75 per cent; damage by wheat stem sawfly was estimated to be about \$6 million in North Dakota alone, in 1944. The larvae of *C. cinctus* feed in the parenchyma and, to a lesser extent, in the vascular tissues of the host plant; vascular bundles are severed at intervals between the nodes during feeding, interfering with translocation of solutes in the phloem to the developing ear, and reducing the yield of grain. A resistant, solid-stemmed wheat variety, aptly named Rescue, was developed in Canada and was released there in 1946. Rescue became extensively grown in areas of Canada and the USA, where serious attacks of stem sawfly had been common. Rescue has continued to give a good control of *C. cinctus* in North America since 1946, and no biotypes of stem sawfly to which Rescue is susceptible have been recorded (Holmes and Peterson, 1957). Rescue has been extensively grown in Montana in recent years and yield losses from stem sawfly attack have been greatly reduced as a result (Luginbill and Knipling, 1969). Populations of *C. cinctus* decreased by more than 90 per cent annually wherever Rescue was grown, and stem sawfly has almost disappeared from certain areas of North America where this variety has been grown for several years. Although Rescue has such outstanding resistance to wheat stem sawfly, it gives lower yields and has poorer grain quality than some sawfly-susceptible wheats in the absence of this pest. For this reason, a rotation of susceptible wheats and Rescue has been advocated in Montana. Luginbill and Knipling (1969) predicted that, if susceptible varieties are grown for three years followed by two years of Rescue, populations of wheat stem sawfly will be kept to an acceptable level.

The degree of resistance to stem sawfly in wheat is related to the pithiness of the stems, which is under genetic control but is strongly influenced by environmental conditions. *C. cinctus* larvae can survive only in wheat stems that are not solid (Holmes and Peterson, 1957). Hybrids between solid-stemmed

parents are almost completely resistant to sawfly, and F_1 generations between solid- and hollow-stemmed wheats show intermediate levels both of stem solidity and resistance to sawfly (McNeal *et al.*, 1971; Wallace, McNeal and Berg, 1973). Solid-stemmed *Triticum durum* lines are also resistant to stem sawfly and have been used as sources of resistance (Tsvetkov, 1973).

CEREAL LEAF BEETLE

Cereal leaf beetle, *Oulema melanopus* (L.), can be an important pest of wheat, barley and oats, particularly in North America. Eggs are laid in large numbers on the leaves of susceptible varieties; the larvae which hatch from the eggs devour the leaves and, if populations of beetles are high, serious losses of grain yield can occur. Schillinger and Gallun (1968), and Ringlund and Everson (1968), showed that pubescent (hairy) wheats are resistant to cereal leaf beetle in the field. The oviposition behaviour of the adult female, the viability of the eggs and the growth of surviving larvae are all adversely affected by pubescence. The females appear reluctant to lay eggs on hairy leaves, which therefore have fewer eggs on

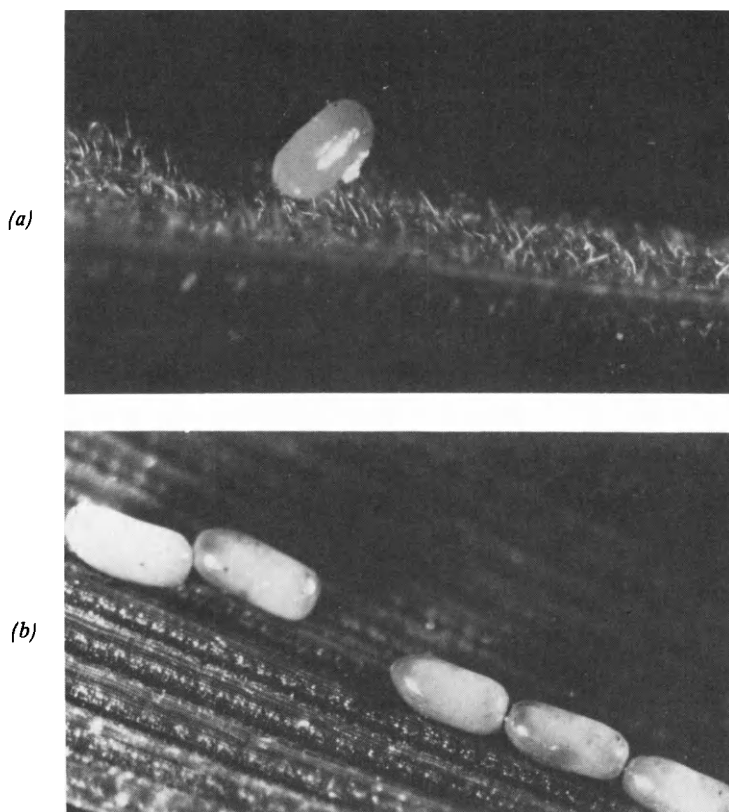


Figure 11.1 Fewer eggs of the cereal leaf beetle (*Oulema melanopus*) are laid on pubescent (hairy) wheat leaves (a) than on glabrous leaves (b). Hatching of eggs on pubescent leaves is also inhibited because the eggs are more liable to desiccation. (By courtesy of Dr P.G. Coleman, UDSA, Michigan State University, East Lansing, USA)

them (Figure 11.1). Eggs seem to be more liable to desiccation on hairy leaves, and less than 10 per cent hatch. Only 20 per cent of those larvae which do hatch on hairy leaves live for more than three days, and the survivors gain weight more slowly on pubescent than on glabrous leaves. Both the density and length of hairs (trichomes) on the leaves and stems seem to be important in conferring resistance (Gallun *et al.*, 1973; Hozie, Wellso and Webster, 1975; Webster *et al.*, 1975; Webster, 1977). Resistance is also associated with waxiness of the leaf surface (Guslitz, 1976).

Schillinger (1969) screened many wheat genotypes for resistance to cereal leaf beetle, and found that one line, CI 8519, is particularly resistant (Figure 11.2). It was the least preferred for oviposition, and only about 10 per cent of *O. melanopus* larvae survived on it for more than a few days. In contrast to the



Figure 11.2 CI 8519, a hard red winter wheat of Russian origin, is resistant to the cereal leaf beetle (*Oulema melanopus*) whereas Genesee, a soft white winter wheat developed in the USA, is very susceptible. (By courtesy of Dr J.A. Webster, USDA, Michigan State University, East Lansing, USA)

situation in wheat, there were no varietal differences in the survival of larvae on the barley and oat genotypes examined by Schillinger (1969). However, there was little oviposition on some barleys, particularly line CI 6671, and on the oat lines, CI 1795 and CI 3393. Tešić, Maksimović and Kuburović (1973) also found that there is no useful antibiosis resistance to cereal leaf beetle in barley or oats.

Long and dense hairs on the leaves and stems may confuse the larvae of *O. melanopus* and impede their movements in much the same way as hairs on pubescent cottons inhibit the movements of first instar larvae of the cotton pink bollworm (*Pectinophora gossypi*) (Smith, Wilson and Wilson, 1975). Pubescence does not appear to encourage parasites of *O. melanopus* larvae because parasite populations have been found to be similar on glabrous and pubescent wheats (Casagrande and Haynes, 1976).

Pubescence and resistance to cereal leaf beetle larvae are dominant characters in *Triticum turgidum*, the presence of hairs being controlled by two or three

major genes; the length of hairs is controlled by a few additive genes (Leisle, 1974). Less is known about the genetics of resistance to *O. melanopus* in bread wheat (*T. aestivum*) but Ringlund and Everson (1968) showed that pubescence is quantitatively inherited and that gene action is additive. In crosses between Genesee (a US glabrous, susceptible variety) and CI 9321 (a Russian pubescent, resistant variety) the F_1 showed an intermediate pubescence. Major resistance genes are carried on chromosome 4A and minor genes on chromosomes 5A, 2A and 7B (Smith and Webster, 1973).

Although knowledge about the mechanisms and genetics of resistance to cereal leaf beetle is incomplete, resistant wheat varieties have given an excellent control of this pest in the field. In field experiments, populations of *O. melanopus* were six times as great throughout the season in plots of a susceptible variety Genesee, as in those of the resistant Veh (Casagrande and Haynes, 1976). There have been no problems with resistance-breaking biotypes of *O. melanopus* and resistant wheat varieties will probably play an increasingly important part in the control of cereal leaf beetle in the future.

CEREAL CYST NEMATODE IN BARLEY

Resistant varieties of small-grain cereals have made contributions to the control of nematode pests, particularly of *Heterodera avenae* (the cereal cyst nematode) and *Ditylenchus dipsaci* (the stem nematode). Severe damage to wheat, oats and barley by *H. avenae* has been reported from many parts of the world including western Europe, the USSR, Canada, India and Australia (Meagher, 1977). The average annual loss caused by *H. avenae* in the UK alone is estimated to exceed £2 million (Cotten, 1970a). Crop rotation is the main control method because, although many effective nematicides are available for the chemical control of cereal cyst nematode, these are generally too expensive for routine use.

Most of the work on breeding for resistance to cereal cyst nematodes has been concerned with barley. Nilsson-Ehle (1920) found that several Scandinavian spring barley varieties, including Abed Rex, Primus and Svantials, were resistant to *H. avenae*, and that this resistance was inherited as a dominant character. It was not until the 1950s, however, that breeding programmes for resistance to *H. avenae* in barley were started in several European countries. Anderson (1961) found that Herta is very susceptible and that Drost is resistant, but only to certain Danish populations. A genotype of *Hordeum pallidum*, designated No. 191, was resistant to populations that attacked Herta and Drost. The populations that could attack Herta but not Drost or *H. pallidum* No. 191, were termed pathotype (race) 1 and those that could attack Herta and Drost but not No. 191 were called pathotype 2. Fiddian and Kimber (1964) studied biotypes of *H. avenae* in England, and found that all the populations tested produced cysts on Proctor, some produced cysts on Drost and none produced cysts on *H. pallidum* No. 191. The biotype situation in England, therefore, seems to be very similar to that in Scandinavia. Most of the *H. avenae* populations from western England were identified as pathotype 1, and most of those from eastern England as pathotype 2. Hayes and Cotten (1971) found some cysts on roots of the hitherto resistant *H. pallidum* No. 191, and nematode populations derived from these cysts were designated pathotype 3. Another

barley variety, Harlan 43, was resistant to this pathotype but was only partially resistant to pathotypes 1 and 2. According to Cook and Williams (1972) there are at least six pathotypes of *H. avenae*, which have been identified in different parts of the world. Pathotypes 1 and 2 of *H. avenae* are widespread in Europe (Videgård, 1974; Saynor, 1975) but pathotype 3 is rare. Several pathotypes are probably present in Australia, including pathotype 3 (Ellis and Brown, 1976).

Cotten and Hayes (1969) and Williams (1970) have studied some of the mechanisms of resistance in *H. pallidum* No. 191 and other resistant barleys. It seems that larvae can invade roots of resistant plants as readily as those of susceptible plants, but the development of mature females is prevented in resistant plants; as a consequence, there are fewer eggs in the soil after crops of resistant varieties than after susceptible crops. The development of larvae is inhibited in resistant barleys, such as Sabarlis, and stops at the third stage although a few individuals, mostly males, may reach maturity (Rivoal, 1976). A high ratio of males to females seems also to be a characteristic of *H. avenae* in resistant wheats (Brown, 1974).

Williams (1970) reported that, in a field experiment, a susceptible barley variety yielded significantly more grain where a resistant variety had been grown in the previous year than after a succession of susceptible crops. Over a period of four years, resistant spring barleys outyielded susceptible varieties in heavily infested soil by an average of about 9 per cent; in uninfested soil, resistant and susceptible varieties gave approximately equal yields of grain (Cotten, 1970b). In this experiment, populations of *H. avenae* declined faster in soils in which resistant plants had been grown than in those with susceptible plants. No change in pathogenicity of *H. avenae* populations was detected in plots where resistant varieties had been grown for three successive years.

Cotten and Hayes (1969) found that, in four resistant barley varieties, resistance to *H. avenae* is controlled by a single dominant gene; the resistance of *H. pallidum* is controlled by the same gene. At least six genes control resistance to *H. avenae* in barley; three of these (Ha , Ha_2 and Ha_3) are probably located on the long arm of chromosome 2 because they are linked with the genes that control the 6-row and ligule-less characters, which are carried on that chromosome. Later work has also indicated that the Ha_2 and Ha_3 genes are on chromosome 2, are closely linked with each other, and may even be alleles at the same locus (Andersen, 1976). In wheat, resistance to pathotype A of *H. avenae* is controlled by a single dominant gene, the dominant allele (Cre) being located on chromosome 2B (Slootmaker *et al.*, 1974).

Resistant varieties may become much more important in the control of cereal cyst nematode in the future. In field experiments in the UK, a resistant spring barley variety, Sabarlis, outyielded susceptible varieties by more than 16 per cent in nematode-infested soil, and populations of *H. avenae* in the soil were significantly reduced for succeeding cereal crops (Graham and Stone, 1975). Resistance to *H. avenae* is race-specific and there is, consequently, a danger that resistance-breaking pathotypes of this pest will become common when resistant varieties become extensively grown. Nevertheless, the benefits of resistant varieties are unlikely to be nullified quickly by such pathotypes because the spread of soil-borne pests is generally slow and new pathotypes would not be disseminated rapidly. However, this does not affect the urgency of finding

alternative sources of non-race-specific or more durable forms of resistance to *H. avenae*. Breeding resistant varieties will probably continue to be a cheap, easy and effective way of controlling this pest where control by an adequate rotation is not possible.

OAT STEM EELWORM

Oat stem eelworm (*Ditylenchus dipsaci*) can be a serious pest of oats in many parts of the world, including western Europe and parts of the USA. Infested oats usually have a characteristic swelling of the leaves and leaf sheaths just above ground level, which gives rise to 'tulip root' symptoms. Infested plants are unthrifty and grain yields are reduced.

Ditylenchus dipsaci is apparently unable to develop to maturity on resistant oat varieties in the field (Goodey, 1937). An old English 'land-race' variety, Grey Winter, and a variety (S8) that was derived from it, are particularly resistant. Later work by Griffiths, Holden and Jones (1957) showed that there is no resistance to penetration of *D. dipsaci* larvae in Grey Winter, but that the development and reproduction of the nematode is impeded in some way. Grey Winter has formed the basis of many breeding programmes for resistance to stem eelworm. Although its resistance is apparently controlled by a single dominant gene, several resistant varieties derived from it have been grown successfully on a large scale for many years. Several methods of testing for resistance to *D. dipsaci* have been reported. For example, Goodey (1937) artificially inoculated field plots with buried dried, infested oat straw. Experimental plots have also been grown on land that is naturally and heavily infested with *D. dipsaci*. Brendler *et al.* (1971) grew plots of eight oat varieties on both infested and fumigated soils, to evaluate resistance to *D. dipsaci* on the basis of grain and straw yield. Similar methods have been used in selecting for resistance to stem eelworm. Laboratory tests, in which seedlings are injected with suspensions of *D. dipsaci*, have also been devised for selecting and testing for resistance.

Griffiths *et al.* (1957) tested a large number of *Avena sativa* varieties and *Avena* spp. for resistance to *D. dipsaci*. Most of the varieties were susceptible but four from Yugoslavia, one from France (Avoine d'Hiver) and one from Ireland (Fluisse) were resistant. Several unnamed Turkish types of *Avena byzantina* were also resistant, as were two types of *A. ludoviciana*.

Resistant oat varieties have been grown in many countries other than the UK, including Sweden, Belgium and the USA. In Belgium a resistant variety, Greta, which was derived from a cross between a resistant British variety (S172) and a local variety, Blanche du Vieux Moulin, has yielded very well under conditions of heavy *D. dipsaci* infestation, but is less productive than many other varieties in uninfested soil (Clamot, 1968). Resistant oat varieties, including Curt, have been recommended for use on nematode-infested soils in California (Brendler *et al.*, 1971).

Although the oat stem nematode has decreased in importance in many parts of the world, mainly because of a greatly reduced area of oats, resistant varieties have already made a very significant contribution to the control of this pest in several countries. No serious problems have been encountered with resistance-breaking biotypes of *D. dipsaci* in oats.

CONCLUSIONS

Most of the work on breeding for resistance to insect and nematode pests of small-grain cereals has been very successful and many resistant varieties have been developed. Some of these have been grown on a large scale, particularly in the USA, and have significantly reduced the amount of pest damage. For example, varieties with resistance to Hessian fly have increased the value of the wheat crop in North America by several millions of dollars annually for nearly fifty years. Stem sawfly is no longer a serious pest of wheat in those parts of Canada and the USA where resistant varieties have been grown for several years. Resistant wheat varieties have also given an excellent control of cereal leaf beetle in North America. Damage by cereal cyst nematode in barley, and by stem eelworm in oats, has been reduced by resistant varieties, particularly in Europe.

Although resistance-breaking biotypes of the Hessian fly, greenbugs and the cereal cyst nematode are known to occur, these have not, so far, been a serious problem. Resistance to all the main pests of cereals has been durable, even when resistant varieties have been grown very extensively. There are, therefore, excellent prospects of achieving a permanent and effective control of many of the most important pests of small-grain cereals by means of resistant varieties. Varieties with multiple resistance to more than one insect pest have been developed; these include wheat varieties with resistance to both stem sawfly and cereal leaf beetle (Wallace, McNeal and Berg, 1974). Thus, resistant cereal varieties may be developed which will each control a wide range of invertebrate pests.

Cotton

Of the very many insect pests that attack cotton, bollworms (which are the caterpillar larvae of several species of moth) are probably the most important. These caterpillars feed in the fruit or bolls and cause very serious damage to the seed and lint, which in turn reduces both yield and quality. There are good prospects that these bollworms will eventually be controlled by resistant varieties.

In the USA, *Anthonomus grandis* (the boll weevil) is probably even more important than bollworms but, until recently, little progress had been reported in breeding for resistance to this pest. However, early rapid-fruiting varieties which escape boll weevil damage have been bred.

In Africa and India, species of jassids (*Empoasca* spp.), which are sap-sucking Hemiptera bugs, are very important pests and resistant varieties have made a considerable contribution to their control. An aphid, *Aphis gossypii*, is also found wherever cotton is grown, but no good sources of resistance to this pest have been reported.

BOLLWORMS

Many species of bollworm attack cotton. The most important of these are *Heliothis zea* (cotton bollworm or corn earworm) and *H. armigera*; *Earias insulana* and *E. biplaga* (spring bollworms); *Diparopsis* spp. (red bollworms) and *Pectinophora* (*Platyedra*) *gossypiella* (pink bollworms).

Cook (1906) observed that some strains of *Gossypium barbadense* are almost immune to *Heliothis zea*, and he suggested that the contents of the pigment glands on the leaves might be responsible for this resistance. No resistant varieties have been widely grown, however, and it is only recently that much attention seems to have been given to breeding for resistance. *Heliothis* spp. have become much more damaging in recent years because many populations have become resistant to organochlorine insecticides; resistant varieties would greatly increase the effectiveness of integrated control measures (Canerday and Baker, 1976).

Resistance to insect pests in cotton has often been attributed to the presence of subepidermal glands which contain high concentrations of a polyphenol pigment, gossypol (e.g. Lukefahr and Martin, 1966; Lukefahr and Houghtaling, 1969). Wilson (1971) found that larvae of *Heliothis virescens* show a marked distaste for cotton plants with many pigment glands or which have a high content of gossypol in the seeds. Glandless Acala cottons are more susceptible to insect pests generally than are those with glands (Benedict *et al.*, 1977). A high density of glands and a high concentration of gossypol are positively correlated with the number of gland-determining alleles present, and with non-preference and antibiosis types of resistance to *H. virescens* larvae (Wilson and Shaver, 1973). Glandless varieties of cotton are more susceptible to *Earias biplaga* than are frego-bract or nectariless varieties (Reed, 1974). The number of glands in cotton is simply inherited and plant breeders can easily select for high gland number and high gossypol content (Wilson, Smith and Remington, 1973). However, gossypol is a very poisonous compound; high gossypol concentrations in seed may be undesirable because cottonseed cake is an important foodstuff for livestock, including pigs and chickens.

The concentration of gossypol and the glanded condition are not the only factors in resistance to bollworms, because only small differences in oviposition or damage by *Heliothis zea* were observed between certain glandless and glanded lines of cotton (Oliver, Maxwell and Jenkins, 1970); in these experiments *H. zea* grew more slowly on the glanded lines but work with *H. virescens* suggests that this inhibition of growth could not be attributed only to a high gossypol content of the flower buds (Shaver, Garcia and Dilday, 1977).

Lukefahr, Houghtaling and Graham (1971) reported that strains of cotton which have glabrous leaves are less satisfactory for oviposition by *Heliothis* spp. than those with hairy leaves — the number of eggs laid on glabrous leaves was sometimes only 20 per cent of that on hairy leaves. Stadelbacher and Scales (1973) also found that hairy cotton plants are preferred for oviposition by *Heliothis* spp.

H. zea and *Pectinophora gossypiella* laid more eggs on pubescent Acala cottons with nectaries than on smooth-leaved, nectariless strains (Davis and Ellington, 1974). Larvae of *P. gossypiella* usually feed only when they encounter leaf nectaries, but a high density of hairs on the stems seems to confuse and impede the movements of first-instar *P. gossypiella* larvae (Smith, Wilson and Wilson, 1975; Wilson and Wilson, 1977). Pubescence therefore seems to contribute to bollworm susceptibility in one respect and to resistance in another. Nectariless cotton lines with high gossypol content are being developed, in the hope that these will show a very high level of resistance to bollworms.

Other varietal differences in resistance to *Pectinophora gossypiella* have been observed. The lint and seed of Pima S4 were much more badly damaged by

P. gossypiella than were those of Pima S3 in field experiments at two centres in India (Kittock and Pinkas, 1971). *P. gossypiella* prefers to oviposit on American *G. hirsutum* varieties than on *G. arboreum* 'desi' varieties from India; one 'desi' variety (AK235) and an American *G. hirsutum* variety expressed antibiosis against the pink bollworm (Muqueem, 1969). Larvae of *P. gossypiella* develop more slowly on several wild strains of primitive cotton from Guatemala and Mexico (Anonymous, 1973) and these may be valuable additional sources of resistance to bollworms. The genetics of bollworm resistance in *G. arboreum*, which is also resistant to *Earias insulana* and *E. vitella* (Butani, 1974), has been studied by Singh (1976).

Several hundred strains of cotton have been evaluated for resistance to *P. gossypiella* in Arizona by incorporating extracts from the bolls in a laboratory diet (Wilson and Wilson, 1975a). Diets produced from the carpel walls of 42 strains significantly inhibited the growth and development of pink bollworm larvae, as did the boll contents of 23 other strains. Extracts from both carpel walls and boll contents from two strains affected the growth and development of *P. gossypiella* larvae. This method may be useful in selecting for resistance to all bollworm species and has already identified potentially useful sources of resistance to the pink bollworm. Wilson and Wilson (1975b) have also shown that segregating populations of cotton can be screened for resistance to *P. gossypiella* by using X-rays to compare the extent of seed damage.

Although breeding for resistance to bollworms has not been one of the most important breeding objectives in cotton, there are good sources of resistance which can be used. Cottons in which the nectariless, glabrous and high-gossypol resistance characters were combined, supported little, if any, increase in *Heliothis zea* and *H. virescens* populations (Lukefahr, Houghtaling and Cruhm, 1975). *Heliothis* larvae were parasitized by a fungus, *Spicaria rileyi*, to a greater extent in an open-canopy cotton variety than in a similar but closed-canopy variety (Burleigh, 1975). This suggests that *Heliothis* may be less damaging in open-canopied cottons generally. Although such cotton varieties could become an important means of controlling bollworms, Wilson and Wilson (1977) consider that the need to apply insecticides will not be entirely eliminated by varieties with resistance based on morphological characters.

BOLL WEEVIL

The cotton boll weevil, *Anthonomus grandis*, which is indigenous in Mexico, first became important in the USA Cotton Belt in the early 1950s when most of the cotton types grown there were green-seeded with small bolls, and had a long fruiting season. These types were particularly susceptible to boll weevil damage and were soon replaced by large-bolled, early-fruiting, white-seeded varieties from the Mexican plateau. These white-seeded varieties were of poorer quality than those which they replaced, but breeders have now managed to combine earliness with high quality (Hutchinson, Silow and Stephens, 1947; Purseglove, 1968). The resistance of the white-seeded varieties is really a pest escape mechanism, because weevil populations increase as the season progresses and early-fruiting cottons are, therefore, not as liable to become infested with large populations.

Other forms of resistance to the boll weevil have recently been found; Maxwell *et al.*, (1969) found that oviposition by *A. grandis* is lower on *Gossypium barbadense* (Egyptian cotton) than on *G. hirsutum* (American cotton). The factor which suppressed oviposition has now been successfully transferred to *G. hirsutum*. Non-preference for oviposition by *A. grandis* is also apparently associated with the 'frego-bract' character, only about one-third as many eggs being laid on plants with frego bracts as on normal bracted varieties (Lincoln *et al.*, 1970). Fewer insecticide applications are needed to control boll weevils on frego-bract varieties as a result of this non-preference for oviposition by *A. grandis* (Jenkins and Parrott, 1972). Attempts are being made to combine resistance factors from *G. barbadense* with this non-preference resistance of frego bract cottons (Jenkins, 1976). The characters frego bract, male sterility and red stem colour each contribute to non-preference resistance to *A. grandis* (Reddy, 1976).

JASSIDS

Jassids (*Empoasca* spp.), which are sap-sucking bugs (Hemiptera), are very serious pests of cotton in Africa and India. Resistant varieties have been extremely important in the control of these pests (Arnold, 1969). The first jassid-resistant varieties were bred in South and East Africa and in the Sudan (MacDonald, Ruston and King, 1945; Knight, 1952). The main source of resistance has been Cambodia cottons from India, which have very hairy leaves and stems. Pubescence (hairiness) is usually associated with resistance to jassids in all cottons, and Upland and Cambodia cottons have been selected for increased hairiness.

Knight (1952) showed that resistance to jassids in perennial types of *G. barbadense* is due to a partially dominant gene H_1 together with several minor modifying genes. Hairiness and jassid resistance are also controlled by a dominant gene in varieties of *G. hirsutum*, *G. tomentosum* and *G. arboreum*. However, Muttuthamby, Aslam and Khan (1969) found that two pairs of complementary genes were involved in the genetic control of hairiness; one pair of genes is present in Pak 51 and L 11 from Pakistan and the other pair occurs in Empire Red Leaf and Acala. Although there is some doubt about the details of the genetic control of hairiness and jassid resistance, it is generally agreed that they are simply inherited, dominant characters which are easy to use in breeding programmes.

Screening for improved jassid resistance has also been carried out in India, where it has been confirmed by Batra and Gupta (1970) that the length of hairs on the lamina and the density of hairs on the midrib and lamina are important in jassid resistance. They found that the thickness of the palisade cells of the leaves is also important, as suggested previously by Yadava, Mital, and Singh (1967); the diameters of the cortex and midvein of the leaves are smaller in resistant than in susceptible cotton varieties.

Several varieties carrying genes for pubescence and resistance to jassids have been widely grown in many of the important cotton-growing areas of Africa, India and the Philippines. For example, a variety, Hirsu-anom (H 59), which was derived from a cross between *G. hirsutum* and *G. anomalum*, is resistant to *Empoasca* spp. and has been recommended for general cultivation

in the Punjab (International Cotton Advisory Committee, 1974). In the Philippines, Baltazar 3 is resistant to *Empoasca bigittula*, presumably because of the dense hairs on its leaves (Al-Azawi and Campos, 1974).

CONCLUSIONS

The excellent resistance to jassids, which has been transferred from Cambodian cottons to many modern cotton varieties, must be considered to be one of the most successful examples of breeding for resistance to insects, in any crop plant. Resistant varieties are now probably the main method of controlling jassids in many cotton-growing areas.

Although resistant varieties have not, so far, made similar contributions to the control of bollworms or boll weevils, good resistance is available for plant breeders to exploit. It is interesting that the length and density of hairs on the leaves of cotton plays such an important but contrasting role in resistance to two important pests; hairiness is associated with resistance to jassids but with susceptibility to oviposition by bollworms, suggesting that it might be difficult to achieve the highest expression of resistance to both jassids and bollworms in a single variety. However, other forms of resistance to bollworms are available, particularly antibiosis associated with the presence of glands on the leaves (Benedict *et al.*, 1977) and pest escape associated with early maturity (Smith and Huffaker, 1973). Higher levels of resistance to the main insect pests are very desirable because all cotton varieties tested in the USA by Canerday and Baker (1976) sustained injury from pest attack, that was above the economic threshold, when insecticides were not used.

Sugar Beet

APHIDS

Differences in heritable resistance to *Myzus persicae* and *Aphis fabae*, which are the principal aphid vectors of sugar beet yellowing viruses (*see* page 239), were first observed by Russell (1960) in a field experiment near Cambridge, England. In this experiment heavy natural infestations of *M. persicae* and *A. fabae* occurred in July, and some inbred sugar beet breeding lines seemed to be less suitable than others for rapid multiplication of either or both species. Plants of some lines suffered less direct damage from aphid feeding than others. These differences between inbred lines were later confirmed in glasshouse experiments (Russell, 1966a). Seedlings were infested with adult apterous aphids and the number of aphids on each seedling was counted at regular intervals for five or six weeks. Large differences in resistance to *M. persicae* and *A. fabae* were observed between inbred lines (*Figure 11.3*) and selections were made for improved resistance. Although varietal differences in aphid preference and tolerance were noted, antibiosis (expressed as resistance to aphid multiplication) was probably the most important type of aphid resistance.

Non-preference (resistance to settling) of *Myzus persicae* is not necessarily associated with non-preference of *A. fabae*, and breeding for resistance to the two species must, therefore, be considered separately. However, screening for resistance to *M. persicae* and *A. fabae* can be carried out simultaneously on the same plant (Russell, 1966b; Lowe, 1972). Resistance to apterae (wingless forms of aphids) is not always associated with resistance to alatae (winged forms) and it is probably necessary to select separately for these two forms. Plants of

varieties and breeding lines with exceptionally low aphid populations in these experiments were selected for seed production and progeny testing. The progenies of some selections were more resistant than the parent plants, and resistance is obviously complex.

Resistance to *M. persicae* and *A. fabae* has not been related to any gross morphological characteristic of the host plant. However, aphids generally settle preferentially on sugar beet plants which contain high concentrations of free

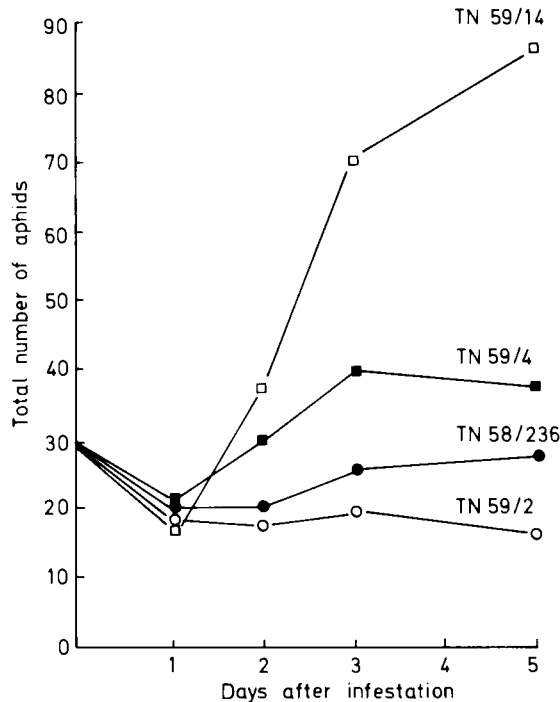


Figure 11.3 Differences between the resistance of four sugar beet inbred lines to *Myzus persicae* in a glasshouse test. (From Russell, 1966a)

sugars (Russell, 1969). Haniotakis and Lange (1974) found varietal differences in the probing behaviour of *M. persicae* on sugar beet using an electronic recording system; such a system could be used to investigate mechanisms of resistance to aphids and in selecting plants for non-preference resistance. Non-preference to aphids is not always associated with resistance to aphid multiplication, although some lines express both forms of resistance, which seem to be independently inherited and controlled by polygenes. Recurrent selection for resistance has resulted in small improvements in each generation. Lowe and Russell (1969) pointed out that there would be two main benefits from aphid resistance in sugar beet: first, direct damage caused by feeding of aphids would be reduced; second, the spread of aphid-transmitted viruses would be decreased and delayed. *A. fabae* is a poor vector of beet yellowing viruses but can cause considerable direct feeding damage; this damage might be significantly reduced in varieties that express antibiosis (resistance to aphid multiplication) and tolerance. *M. persicae*, however, is mainly important as a vector of yellowing viruses, and even small populations within a crop are undesirable; tolerance

to *M. persicae* is, therefore, unlikely to be a useful breeding objective. A combination of non-preference and antibiosis resistance might give a good control of *M. persicae* in the field. However, non-preference could, under certain conditions, cause increased virus spread because aphids might move more often from one plant to another in crops of a 'non-preferred' variety, thereby spreading viruses more quickly. For this reason, Lowe (1974a) has concentrated on screening mainly for improved antibiosis within breeding lines; plants are each infested with a predetermined number of adult aphids and the size of aphid population on each plant is assessed at intervals. Plants supporting small aphid populations are selected for seed production and further breeding.

Although most of the experimental and selection work on aphid resistance in sugar beet has been carried out in the glasshouse, Lowe (1971) showed that differences in aphid resistance can be demonstrated successfully in the field. For example, a breeding line, VT 90, has been consistently more resistant to *M. persicae* than many others, in both glasshouse and field tests. The incidence of virus yellows is also usually much lower in field plots of aphid-resistant varieties than in those of susceptible varieties under conditions of natural aphid infestation. Large differences exist between the colonizing ability and overall vigour of individual aphid clones, but resistant varieties have generally supported smaller aphid populations in field and glasshouse tests, irrespective of the aphid clones involved (Lowe, 1974b). Resistance to aphids in sugar beet is, therefore, probably non-race-specific and resistance-breaking biotypes are unlikely to be a problem. The expression of resistance to aphids in sugar beet is very labile and can be affected by environmental factors, including the availability of certain nutrients to the host plants.

It is unlikely that resistance to aphids alone can give an adequate control of virus yellows; it is, therefore, desirable to combine resistance to aphids with resistance to virus infection and virus tolerance when breeding new sugar beet varieties (see page 241). Maris Vanguard, a virus-resistant multigerm variety, which was first marketed in eastern England in 1965, has shown a high level of virus tolerance, resistance to infection with yellowing viruses, and some resistance to *M. persicae*. Experimental monogerm lines, such as VT 95, which express resistance to aphids and to virus yellows, have been used in the breeding of virus-resistant monogerm varieties (Howard *et al.*, 1970).

Although only small, quantitative differences in heritable resistance to *M. persicae* and *A. fabae* have been discovered so far, such differences can contribute substantially to the control of aphids and aphid-transmitted viruses in the field (Lowe, 1972; 1975). Predators are able to give a better control of aphids in aphid-resistant varieties than in more susceptible varieties. This implies that there can be a valuable synergistic effect on the control of aphids when heritable resistance is combined with other methods of control.

BEET EELWORM OR CYST NEMATODE

Heterodera schachtii is a major pest of sugar beet, particularly in Europe and the USA. A strict control of crop rotation, which restricts the growing of susceptible crops to once every four or five years, has been successful in several countries in delaying the spread of *H. schachtii* and in reducing damage (Dunning and Dyke, 1977). Nematicides, particularly aldicarb, have given

an effective control of *H. schachtii* in field experiments but these chemicals are expensive, have high mammalian toxicity and often give only a transient control. Varieties resistant to the beet cyst nematode are, therefore, still urgently needed to augment other control measures. Programmes of breeding for resistance were started in several countries, including the USA, UK, the Netherlands and Poland, during the 1950s.

Hijner (1951) working in the Netherlands, reported that three species in the genus *Beta* are highly resistant to *H. schachtii*: these are *B. patellaris*, *B. procumbens* and *B. webbiana*. These results were later confirmed by several other workers. Steele and Savitsky (1962) found that, although these species are virtually immune to *H. schachtii*, single female nematodes can develop on a few plants of *B. patellaris*; the presence of such females on resistant plants may indicate the existence of resistance-breaking biotypes of the pest (Shepherd, 1959). Savitsky and Price (1965) crossed these wild resistant species with susceptible sugar beet lines (*B. vulgaris*) and found that the resistance in each species is controlled by a single dominant gene (Savitsky, 1976). The chromosome carrying this resistance gene in *B. procumbens* has been transferred to *B. vulgaris* by producing hybrid trisomics containing 18 *B. vulgaris* chromosomes and one chromosome from *B. procumbens*. However, segments of this alien chromosome were progressively lost in succeeding generations and this was associated with a progressive loss of resistance to *H. schachtii* (Savitsky, 1976). It is a long and difficult process, therefore, to transfer nematode resistance from wild *Beta* species into a genetically stable *B. vulgaris* genotype without losing the nematode resistance, and without also transferring many deleterious characteristics from the wild parent. For these reasons, no successful sugar beet varieties have yet been developed from interspecific crosses.

An alternative approach has been to seek resistance to *H. schachtii* within sugar beet varieties. No immune *B. vulgaris* plants have been found, in spite of very extensive searches by many workers including Shepherd (1958), Price (1966), Jorgenson and Smith (1966), Curtis (1970) and Pawelska-Kozińska and Szota (1970). Attempts have been made to select and breed from sugar beet plants on whose roots relatively few cysts (mature female nematodes) develop; heritable differences in this form of resistance have been reported, but these differences are generally small and easily influenced by changes in environmental conditions. Curtis (1970) concluded from his experiments that varieties with a very high level of resistance to cyst formation are unlikely to be developed from existing varieties. However, varietal differences in the extent of invasion of the roots by *H. schachtii* larvae have been recorded (Korol'chuk *et al.*, 1971), and this kind of resistance should be investigated more intensively.

Heijbroek (1977) has found partial resistance in sugar beet and *Beta vulgaris* s.sp. *maritima* that is based on an unequal ratio of male to female nematodes in resistant plants. The penetration and development of *H. schachtii* larvae are otherwise completely normal in resistant plants. This resistance appears to be polygenically controlled and recessive, and is therefore difficult to use in a breeding programme. The expression of resistance, even in the most resistant *B. maritima* plants, is much less than that in *B. patellaris*, *B. procumbens* and *B. webbiana*.

Results of selecting for tolerance to *H. schachtii* have been more encouraging. For example, Price (1966) found that a popular variety, US41, lost 30 per cent of its potential root yield when grown in nematode-infested soil in California,

compared with only about one per cent loss in a tolerant line. Jorgenson and Smith (1966) reported that 139 lines and varieties out of 750 tested in nematode-infested soil showed some tolerance. Curtis (1970) and Pawelska-Kozińska and Szota (1970) have also reported polygenically inherited differences in tolerance to beet eelworm. Berbeč (1973) noted that the largest roots were often found in heavily infested soil and concluded that it should be possible to select effectively for tolerance to *H. schachtii* in such conditions. Although no resistant or tolerant varieties have yet been marketed, there are good indications that such varieties will eventually be produced. In the USA, a sugar beet line, Acc 107, which was developed from plants selected for nematode resistance, has given acceptable yields in heavily infested soils, under many different water and fertilizer regimes, and supports fewer *H. schachtii* cysts than unselected lines (Whitney and Doney, 1973). Such material should form the basis of tolerant varieties which will greatly reduce damage caused by cyst nematode.

Rice

More than 100 species of insects are pests of rice (*Oryza sativa*) but only about 20 of these are of major economic significance. The most damaging pests are various species of stem borers, leafhoppers and planthoppers. Resistant rice varieties have helped greatly to reduce the amount of pest damage (Pathak, 1972).

RICE STEM BORERS

Rice stem borers are the most important pests of rice in Asia and, although they can usually be adequately controlled by insecticides, a major effort is being made to breed resistant rice varieties in many countries. Pathak *et al.*, (1971) have written a comprehensive account of the work that has been carried out at the International Rice Research Institute (IRRI) in the Philippines.

Stem borers are larvae of several species of Lepidoptera, which burrow into the stems and leaf sheaths of rice plants in which they feed, causing considerable losses of grain yield. Probably the most damaging stem borer is *Chilo suppressalis*, which is often called the striped stem borer or Asiatic rice borer. Panicles of infested tillers may produce no grain or only shrivelled grain, and severely infested tillers may be completely killed. The insect pupates inside the stem from which the moths finally emerge; the adult females lay large numbers of eggs on the laminae or the leaf sheaths of rice plants, and these eggs hatch out to produce the stem-boring larvae.

Varietal differences in susceptibility to stem borers in rice were first recorded in 1917 by workers in Japan. Subsequent work showed that native Japanese varieties were generally damaged less by stem borers than introduced varieties, and that 'unbearded' varieties were more resistant than 'bearded' varieties; the diameter of the stalk was also considered to be an important factor in resistance.

Many thousands of varieties and lines of rice were tested for resistance to stem borers during the 1950s and 1960s in the Philippines and highly significant differences were found between them (*Figure 11.4*). Tests were carried

out in the field out of season when there were few alternative sites for oviposition by adult female moths; this technique resulted in a heavy natural infestation of the experimental plants and varieties which suffered the least damage from stem borers could be selected. Varieties selected in these tests were subsequently re-evaluated for resistance in the field; there was generally good agreement between the results of successive tests.



Figure 11.4 Rice varieties differ in resistance to the yellow stem borer (*Tryporyza incertulas*). In the photograph, alternate pairs of rows are (from left to right) of a resistant variety, IR 1820-52-2, and a very susceptible variety Rexoro. (By courtesy of Dr M.D. Pathak, International Rice Research Institute, Philippines)

Field tests have often been augmented by glasshouse tests in which plants are each infested with ten first-instar stem borer larvae. On many resistant varieties, (e.g. Pai Mang JH) fewer than 10 per cent of the larvae survived and none of these pupated successfully. On susceptible varieties (e.g. Pannai) more than 40 per cent of the larvae survived and about 70 per cent of these pupated. The resistance of most varieties seems mainly to be associated with the reluctance of female moths to oviposit on them. Although some resistant varieties, for example TKM6, have very hairy leaves, hairs do not seem to be of overriding importance in discouraging oviposition. Larvae often grow more slowly and suffer a higher mortality on resistant than on susceptible plants, suggesting that there is an antibiotic factor in resistant plants (Pathak *et al.*, 1971; Oliver and Gifford, 1975); Taitung and TKM6 exhibit a high level of this type of resistance (Das, 1976a). The antibiosis of TKM6 is apparently based on anatomical and morphological features of the host and that of Taitung 16 on a water-soluble biochemical factor. Varieties that are non-preferred by *C. suppressalis* for oviposition do not necessarily also express antibiosis resistance to stem borers. For example, in field experiments in Korea, oviposition was lowest

in Suveon 240 whereas reduction in larval weight gain and pupation rate were lowest in IR747 (Lee, Park and Kim, 1974). Resistance in some varieties is associated with thick layers of sclerenchymatous and lignified tissues in the stem (Patanakamjorn and Pathak, 1967) or with high concentrations of silica (Sasamoto, 1961; Djamin and Pathak, 1967). A high silica content in the host plant may interfere with the feeding of stem borers by wearing away their mouthparts.

Some rice varieties (e.g. Balilla) are reported to express both tolerance and antibiosis to *C. suppressalis* (Galichet, Poitout and Loma, 1976). Others tend to avoid being damaged as much as other varieties by stem borers because they mature either very early or very late in the growing season (Oliver, Gifford and Trahan, 1972) or are tolerant (Das, 1976b). It appears, therefore, that resistance to stem borers in rice can involve many different types of resistance mechanisms, including pest avoidance, reduced oviposition, antibiosis, mechanical resistance and tolerance. It is not surprising, therefore, that the genetics of resistance is not fully understood. However, antibiosis to *C. suppressalis* in rice is controlled monogenically or oligogenically (Athwal and Pathak, 1972). Tolerance, as measured by the amount of stem borer damage in the field, is apparently controlled polygenically.

Varieties resistant to stem borers are often unexpectedly susceptible to other insect pests and to viruses and bacterial diseases, or are low-yielding or low-quality types. For these reasons, selected borer-resistant rice varieties have been included in hybridization programmes with the objective of combining resistance to stem borers with other desirable characteristics. Fortunately, varieties resistant to one species of Noctuid or Pyralid stem borers are usually resistant also to other stem borers (Das, 1976b). A few varieties have multiple pest and disease resistance. For example, although TKM6 is a variety of poor agronomic type, it has surpassed most other varieties in resistance to *C. suppressalis* (stem borer), *Xanthomonas oryzae* (bacterial leaf blight) and *Nilaparvata lugens* (a planthopper). Some progenies derived from crosses between TKM6 and Taitung 16 have shown good resistance to stem borers as well as good agronomic characters (Pathak, 1969).

LEAFHOPPERS AND PLANTHOPPERS

Two of the most important pests of rice in tropical countries are the brown planthopper, *Nilaparvata lugens*, and the green leafhopper, *Nephotettix virescens* (*N. impicticeps*). Light infestations of these insects can reduce plant vigour and yield of grain but heavy infestations can cause loss of entire crops. Perhaps of even greater economic importance than their direct feeding damage, however, is their role as vectors of viruses and mycoplasmas. *N. virescens* transmits several rice viruses, the most damaging of which is probably tungro virus (see page 247); *N. lugens* transmits the grassy stunt pathogen (see page 206). Insecticides are the main control method at present but there are good prospects of breeding for resistance to both these insect pests (Pathak, Cheng and Fortuno, 1969). Work on breeding for resistance to *N. virescens* and *N. lugens* is being undertaken in many parts of the world but particularly at IRRI. Another leafhopper species, *Sogatodes oryzicolus*, is an important vector of hoja blanca virus in South and Central America (see page 246).

About 1000 varieties that had been selected as showing some resistance to stem borers were screened for resistance to *N. virescens* and *N. lugens* in seedling tests at IRRI. Batches of seedlings were grown in cages and exposed to heavy infestations of either pest; the degree of infestation was manipulated so that about 90 per cent of the plants of susceptible varieties were killed. The resistance of varieties which showed good survival under these conditions was later retested by infesting individual plants with a known number of insects and following the development of insect populations on them. In these tests, an indica type, Mudgo, was outstandingly resistant to *N. lugens* and even more highly resistant varieties have since been identified (Pathak *et al.*, 1969; Athwal *et al.*, 1971; Kulshreshtha, Anjaneyulu and Padmanabhan, 1974; Gunawardena *et al.*, 1975); these varieties can give an excellent control of planthoppers in the field (Figure 11.5).



Figure 11.5 The rice variety IR 2061 (right) is much more resistant than IR8 (left) to the brown planthopper (*Nilaparvata lugens*). (By courtesy of Dr M.D. Pathak, International Rice Research Institute, Philippines)

Although tolerance may be implicated in the resistance of some varieties, it appears that non-preference and antibiosis are probably much more important. Larvae of *N. lugens* generally died much more quickly on Mudgo than on a susceptible variety, Pankhari 203. Pathak (1970) considered that plants of Mudgo either lack a factor that stimulates feeding of *N. lugens* or contain a substance that strongly repels this insect. Song, Choi and Bak (1972) reported that *N. lugens* prefers to oviposit on some rice varieties rather than on others, and also that *N. lugens* nymphs grow more slowly and die earlier on resistant varieties. Hirao and Todoroki (1976) consider that non-preference is a more important component of resistance to *N. lugens* than tolerance or antibiosis in Mudgo, particularly in adult plants. Sogawa and Pathak (1970) showed that leaves of resistant plants contain lower concentrations of certain free amino acids, particularly asparagine, than susceptible plants, and that asparagine and sucrose strongly stimulate feeding of *N. lugens* in artificial feeding tests. Part

of the resistance of Mudgo to the brown planthopper may, therefore, be due to an unusually low asparagine content in the leaves.

Resistance to *N. lugens* is controlled in some rice varieties (e.g. Mudgo) by a single dominant gene *Bph*₁ (Athwal *et al.*, 1971; Martinez and Khush, 1974; Chang, 1975). In other varieties (e.g. ASD7) resistance is controlled by a single recessive gene (*bph*₂) which is closely linked or allelic to *Bph*₁. TKM6 is homozygous for *Bph*₁.

The variety Mudgo has shown good resistance to many *N. lugens* populations collected from different parts of the Philippines and has also been highly resistant in naturally infested field experiments in India, Korea and Taiwan. However, a number of biotypes of *N. lugens* have been identified recently in Taiwan and the Philippines, which seem to interact with particular resistance genes in rice varieties. For example, plants with the Mudgo type of resistance (gene *Bph*₁) and the ASD7 type of resistance (gene *bph*₂) have been severely attacked and damaged by 'biotype 11' of *N. lugens* in Taiwan (Cheng, 1975). Many previously resistant varieties, for example IR26, IR28 and IR30 (all of which carry the resistance gene *Bph*₁) and IR36 (with gene *bph*₂), have been attacked by 'biotype 2' of *N. lugens* in the Philippines (Feuer, 1976) and in India and Sri Lanka (Lakshminarayana and Khush, 1977). Resistance to *N. lugens* that is controlled by the major genes *Bph*₁ and *bph*₂ is therefore race-specific and more durable forms of resistance are urgently needed. Two new genes for resistance to the brown leafhopper have recently been discovered, *Bph*₃ from Rathu Meenati and *bph*₄, which is non-allelic and independent of *bph*₂ (Lakshminarayana and Khush, 1977). Unlike the *Bph*₁ and *bph*₂ resistance genes, which cannot easily be combined in the same variety, the two new genes can be used in combination. The resistance of varieties with multiple resistance genes may be more durable than resistance controlled by single genes.

Several rice varieties including Pankhari 203, ASD7 and IR8, are resistant to the green leafhopper *Nephotettix virescens* (Cheng and Pathak, 1972). When first-instar nymphs were caged on resistant plants, about 90 per cent of them died within a few days, compared with about 10 per cent on susceptible plants; the surviving nymphs grew more slowly on resistant plants. Adult *N. virescens* also had a shorter lifespan and laid fewer eggs on many resistant varieties than on susceptible varieties. Although Krishnaiah (1975) found that *N. virescens* oviposited and fed on resistant and susceptible plants to equal extents in his experiments, he found that most larvae did not complete their development on resistant plants. Resistance to *N. virescens* in Pankhari 203, ASD7 and IR8 is controlled by three different, non-allelic dominant genes which are independent of the genes for resistance to *N. lugens* in Mudgo (Athwal and Pathak, 1972). Resistance genes have been identified at five loci in other varieties, including two alleles at the *Glh*₁ locus, two independent single genes (*Glh*₂ and *Glh*₅, an allelic series at the *Glh*₃ locus and a single recessive gene, *glh*₄ (Siwi and Khush, 1977). No reports of large-scale damage by resistance-breaking biotypes of *N. virescens* have been published.

Several rice varieties, including Mudgo and IR5 are resistant to hoja blanca virus and to its principal leafhopper vector, *Sogatodes oryzae* (Galvez, 1971). Resistance to the virus is inherited independently of resistance to the vector, because some varieties are resistant to either *S. oryzae* or hoja blanca virus but not to both. For example, IR8 and TKM6 are resistant only to the leafhopper vector.

CONCLUSIONS

Rice varieties that are resistant to the brown leafhopper are grown over a wide area of Asia and have usually given such a good control of this pest that insecticide applications have been unnecessary (Pathak, 1977). However, resistance-breaking biotypes have severely damaged some resistant varieties and these biotypes are, therefore, a serious potential threat to rice production in many parts of Asia. Resistance to all known biotypes of *N. lugens* is now being sought by plant breeders. The use of mixtures of different rice varieties may help to reduce damage by the brown planthopper (Weerapat, Purivirojkul and Chaturonrangsri, 1977).

Varieties with good resistance to *Nephotettix virescens* and *Sogatodes oryzae* have been identified and these are contributing to the control of these pests. Resistance-breaking biotypes of *N. virescens* and *S. oryzae* have not, so far, been a major problem.

Good progress has been made with breeding for resistance to stem borers and leafhoppers, and many high-yielding, early-maturing varieties have been developed. Several of these varieties have multiple resistance to pests and diseases, although resistance to one insect pest is not necessarily associated with resistance to others (Choi, 1971). Rice varieties have been bred in the Philippines with resistance to bacterial blight, brown leafhoppers and stem borers (derived from TKM6), resistance to blast disease, tungro virus and green planthoppers (from Gam Pai 15) and resistance to the grassy stunt pathogen (from *Oryza nivale*) (International Rice Research Institute, 1975). An Indian variety is resistant to several insect pests, including *Nephotettix virescens*, *N. nigropictus* and *Nilaparvata lugens*, and is also tolerant to bacterial blight and tungro virus (Prakasa Rao and Sastry, 1975).

Maize (Corn)

Maize (*Zea mays*), which is usually referred to as 'corn' in North America, can be attacked by at least fifty species of insect pests. Although some of these can be partly controlled by insecticides, attempts are being made to breed for resistance to most of the important pests, including the European corn borer, the corn earworm and the corn leaf aphid.

EUROPEAN CORN BORER

The European corn borer, *Ostrinia (Pyrausta) nubilalis*, is not an important pest of corn in Europe where it is indigenous. This insect was accidentally introduced into the USA in 1917 and has since become one of the most destructive pests of maize in North America. Although damage by the corn borer has been reduced in recent years by cultural and chemical control methods, considerable losses of yield still occur and the development of resistant varieties is an important breeding objective.

There are two generations (broods) of corn borer each season, and the biological relationship between *O. nubilalis* and its maize host differs for each brood, the first brood attacking young plants, and the second brood affecting the

developing grain. This difference has considerably complicated the work of selecting and testing for resistance in developing new resistant varieties. Nevertheless, more than 40 resistant lines and hybrids were released for use in the US Corn Belt during the 1940s and 1950s by plant breeders in Minnesota and Iowa (Everett, Chiang and Hibbs, 1958; Penny and Dicke, 1959). The many other resistant varieties which have subsequently been bred and grown extensively have greatly reduced damage by corn borers.

Current hybrid maize varieties in the USA differ greatly in their ability to yield well under moderate to heavy infestations of *O. nubilalis*; however, all can become attacked, although to different extents, at the sheath and collar stage (Brindley *et al.*, 1975). Varieties resistant to corn borers in maize have been developed in many countries, including Romania (Mustea, Perju and Cabulea, 1975), the USSR (Susidko, Kokot and Chastiř, 1972) and the USA (Pesho, Dicke and Russell, 1965). One variety from the USA (CI 31A) has shown good resistance to *O. nubilalis* in several major maize-growing areas, including Romania, Canada, the USA, Yugoslavia, Hungary and the USSR (Mustea *et al.*, 1975).

Although naturally infested field plots have sometimes been used in testing and selecting for resistance to the European corn borer, field experiments have generally been infested artificially with egg masses of *O. nubilalis*. These egg masses are produced in the laboratory by rearing very large numbers of insects on artificial diets in plastic dishes, thus producing many millions of eggs annually (Guthrie, Huggans and Chatterji, 1970; Brindley *et al.*, 1975). The best way of inoculating field plants is to place three egg masses in the whorl of each plant (Showers and Reed, 1972). The extent of feeding damage by first and second-brood *O. nubilalis* to the leaves, sheath and collar of each plant can then be compared (Shehata, Hargraves and Davis, 1975).

Increased mortality of second-brood larvae is an important factor in resistance but this can be determined accurately only by dissecting plants at intervals after the eggs hatch; most feeding occurs near the collar and behind the sheath. Large varietal differences in larval mortality have been observed. For example, more than 95 per cent of *O. nubilalis* larvae died within a few days on plants of a resistant inbred, B52, whereas most larvae survived on susceptible plants (Guthrie *et al.*, 1970). Line B52 is resistant to both broods of European corn borer and its resistance has been successfully transferred to sweet corn, in which resistance to the second brood is particularly important (Pounders *et al.*, 1975). However, severe infestations of *O. nubilalis* can seriously impede the development of the primary ear, even in Line B 52 and other resistant lines, and higher levels of resistance are required. Chiang (1968) compared the survival and development of *O. nubilalis* larvae on varieties that were widely grown in 1955 with that on popular varieties of 1965. He concluded that, as a result of selection for resistance to European corn borer, there had been an increase in the general level of resistance of between 5 and 15 per cent during the ten-year period.

There is conflicting evidence concerning the inheritance of resistance to *O. nubilalis*. For example, Penny and Dicke (1956) found that two or three genes, with no appreciable degree of dominance, seemed to be involved in resistance as assessed by the amount of feeding. In later experiments with F_2 and back-cross progenies from crosses between susceptible and resistant lines, resistance seemed to be controlled by a single dominant gene (Penny and Dicke, 1957). However other reports have suggested that the inheritance of resistance to the European corn borer is quantitative and that several genes are involved (Scott, Hallauer and Dicke, 1964). Additive gene effects are important in resistance

to leaf feeding by first-brood larvae, and only one group of genes showed dominance in a diallel-cross experiment involving four resistant and four susceptible inbreds; narrow sense heritability was estimated at 40 per cent (Chiang and Hudon, 1973). When the number of cavities per stalk caused by second-brood larvae was used as a criterion of resistance, resistance was dominant in crosses between resistant and susceptible inbreds (Jennings *et al.*, 1974); additive genetic variance estimates were generally low and epistasis was detected in three of four such crosses. In a diallel cross involving 10 resistant and susceptible inbreds, Jennings, Russell and Guthrie (1974) found that general and specific combining ability for resistance to first and second broods were highly significant, and that resistance to each brood was controlled by different genes. Susceptibility to feeding by *O. nubilalis* is increased by the presence of Tcms (Texas) cytoplasm (Pesho, Russell and Dicke, 1969), but male-sterile plants with Tcms cytoplasm are more resistant to first-brood larvae than are fertile plants (Fomin, 1970). These results show that the expression of resistance to European corn borer can be influenced by cytoplasmic genes.

These often conflicting results concerning the inheritance of resistance to the European corn borer suggest that resistance is complex and may involve several independently inherited mechanisms. There is little or no correlation between resistance of individual plants to the first and second broods of *O. nubilalis* in the field (Pesho, Dicke and Russell, 1965), indicating that resistance mechanisms which operate in young plants (when the first brood of larvae attack), are different from those which are effective in older plants, when second-generation larvae attack. Andrew and Carlson (1976) found major differences between the numbers of egg masses of *O. nubilalis* that were laid on six homozygous sweetcorn and popcorn lines in the field. Sweet, hairless, early-maturing lines had fewer egg masses per plant than pubescent, late-maturing lines. Varietal differences in feeding deterrence of first-brood larvae have also been reported (Scriber *et al.*, 1975). Non-preference may therefore be an important component of resistance.

Beck (1957) found that three chemical resistance factors are involved in resistance to European corn borer; the concentration of these compounds was well correlated with the degree of resistance in five inbred lines of corn, but the tassels of all lines were equally susceptible. The first compound (Factor A) was thought to be important only in young plants, and probably affects the first brood of *O. nubilalis*. The second and third compounds (Factors B and C) contributed equally to the resistance of the internodes, leaf sheaths, husks and silk tissues to the second brood. Loomis, Beck and Stauffer (1957) identified the ether-soluble resistance Factor A as 6-methoxy-2(3)-benzoxazolinone, which occurs in higher concentrations in the leaves than the stems and inhibits corn borer larvae. Factor C has been identified by Klun *et al.* (1970) as 2,4-dihydroxy-7-methoxy-2H-1,4-benzoxazin-3(4H)-one, which is often referred to as DIMBOA. There are significant varietal differences in concentrations of DIMBOA in whorl tissue and these are associated with differences in resistance to *O. nubilalis* to which DIMBOA is distasteful. A high content of DIMBOA in the whorls, which is inherited as a dominant character, has been used as a selection criterion in breeding for resistance to the European corn borer (Russell *et al.*, 1975; Hudon and Chiang, 1977). However, factors other than DIMBOA content are involved in resistance because resistant varieties do not always contain a very high concentration of DIMBOA. Guthrie and Walter (1961) tested 50 inbred lines for resistance both to the corn earworm (*Heliothis zea*) and to

O. nubilalis. No association was found between resistance to these two pests, although some lines were resistant to both insects. This shows that antibiotic resistance factors such as DIMBOA which are effective against *O. nubilalis* are specific in their action.

Several field corn and sweet corn hybrids and lines with good resistance to the European corn borer have been developed in the USA. For example, B 75, a yellow dent line with high resistance to leaf feeding by first-brood *O. nubilalis* larvae is resistant also to several fungal diseases, including southern corn leaf blight (Russell, 1976). The resistance of line B 52 to both broods of European corn borer has already been mentioned. Parasites of *O. nubilalis*, particularly *Nosema pyrausta* (a Protozoan), give a much better control of corn borers on resistant lines such as B 52 than on more susceptible varieties (Lewis and Lynch, 1976); this synergism between control by resistant varieties and control by parasites can greatly reduce corn borer damage. Nevertheless, higher levels of resistance than that expressed by line B 52 are clearly desirable to reduce still further the damage that can occur in severe infestations.

MAIZE STEM BORERS

Chilo zonellus and *C. partellus* are widespread and damaging pests of corn, sorghum, millet and rice in the lowland areas of India and Pakistan and in parts of East Africa. The borers are moth larvae of the Pyralidae family. The female moths lay eggs on suitable host plants, particularly on the leaves, and these hatch about a week later giving rise to stem-boring larvae which burrow into the stem and leaf sheaths of the host plant. Heavy infestations can cause considerable losses of grain yield.

In Pakistan, Khan and Haque (1970) tested a number of locally adapted and foreign inbred varieties for resistance to *C. zonellus* under field conditions and found considerable differences in resistance between lines and varieties; in their tests, the variety Ohio was particularly resistant. Kalode and Pant (1967) also found large differences in resistance to *C. zonellus* between varieties in field tests in India. For example, Ganga Hybrid 101 was more resistant than Patiala Local. Resistant varieties generally had an unusually high content of aspartic acid and low contents of nitrogen or sugars.

One hundred Indian maize lines were screened for resistance to *C. zonellus* in artificially infested field trials, and large varietal differences were found (Chatterji *et al.*, 1971; 1972). In cages, several varieties including Basic Local, A2 and Antigua Gr 1, were preferred for oviposition by *C. zonellus*; some varieties also expressed antibiosis (Sharma and Chatterji, 1971). Mathur and Jain (1972) found that antibiosis is an important component of resistance in several maize varieties; most *C. zonellus* larvae died within 10 days when they were fed on the tissues of resistant varieties, whereas most survived on susceptible plants. More than 140 Indian maize varieties and lines were similarly screened for resistance to *C. partellus* with plants artificially infested at the mid-whorl stage. Several varieties and lines, including Antigua Gr 1, were much more resistant to this pest than the other varieties that were examined.

CORN EARWORMS

Corn earworms (or bollworms) are larvae of moths (*Heliothis zea* in the New World and *H. armigera* in the Old World), which can attack a wide range of crops, including maize, sorghum and cotton (see page 334). Female moths

lay eggs singly on the stems or young grains, and these eggs hatch into caterpillars which feed on the developing grain. Damage to maize can be extensive and the production of sweet corn in parts of the southern USA was stopped for many years because of recurring damage by *H. zea*; the development of resistant varieties in the early 1940s enabled production to be resumed in these areas. Several field corn hybrids, which are partially resistant to *H. zea*, have been developed in the USA and these hybrid varieties are now being widely grown. However, it is often necessary to treat even resistant varieties with insecticides to obtain high yields (Wene, Blanchard and Walter, 1953; McMillian *et al.*, 1972).

Testing and selecting for resistance to *H. zea* has mainly been carried out in field experiments in which first-instar larvae are placed on each plant. Straub, Fairchild and Keaster (1973) and Straub, Fairchild and Zuber (1973), found that *H. zea* larvae grow very slowly on resistant plants and often do not complete their development on some resistant varieties, such as Zapalote Chico. The antibiosis resistance of this variety has been transferred to other maize varieties, although resistance is quantitatively inherited. Widstrom and McMillian (1973) studied the inheritance of resistance to *H. zea* in 11 sweet corn and 11 dent inbreds; additive gene and dominance effects were very important in the sweet corn group but not in the dent lines. These results suggest that antibiosis is controlled by several genes.

Some maize hybrids, for example Dixie 18 and X81-1, are tolerant to *H. zea* because, although they can become heavily infested with earworm larvae in the field, they suffer little damage as a result (Wiseman, McMillian and Widstrom, 1972).

Although the morphological and physiological basis of resistance to *Heliothis* spp. is not understood, several independent factors are probably involved. McCulloch (1920) considered that the attraction of *H. zea* to corn silk, and the ability to discriminate between varieties, was partly an olfactory response. He also found that more eggs were laid on plants with glabrous leaves than on those with hairy leaves. The first generation of corn earworm caterpillars feed in the whorl tissue and the second in the ears; resistance to each generation of *H. zea* was shown by Painter and Brunson (1940) to be independently inherited and different resistance mechanisms are probably involved. The tightness of the husk is apparently an important factor in resistance, the depth of penetration of *H. zea* larvae being much greater in susceptible plants than in resistant plants (Leveck, 1967). The presence, in resistant plants, of substances that are distasteful or toxic to this pest, cannot be ruled out because larvae grow very slowly or die on excised silks of resistant plants (Wiseman, McMillian and Widstrom, 1977). Higher levels of resistance to *H. zea* than those of current varieties would be an advantage because less insecticide would need to be applied in order to achieve economic yields. There is considerable scope for improving the levels of resistance to corn earworms, particularly in the sweet corn groups (Widstrom and McMillian, 1973; Widstrom, Wiseman and McMillian, 1975).

Very little attention seems to have been paid to breeding for resistance to *H. armigera*, but the production of resistant varieties would probably not be difficult.

CORN LEAF APHID

The corn leaf aphid (*Rhopalosiphum maidis*) is widely distributed throughout the world in temperate and tropical regions, where it is a pest of several crop

species including maize, sorghum and sugar cane. It is present in the North American Corn Belt in most years and, when conditions are favourable for the rapid multiplication of aphids, *R. maidis* can cause severe damage to the maize crop. During the 1920s and 1930s, differences in resistance to *R. maidis* were observed between many inbred lines and hybrids in naturally infested field trials, and many partially resistant varieties have been developed in the USA.

Testing and selection for resistance to *R. maidis* must be carried out in the field with adult plants, because this aphid is apparently not able to survive on corn seedlings. The reasons for this are not understood, but neither plants of a resistant hybrid, K1859, nor those of a susceptible hybrid, K2234, supported larvae of *R. maidis* until the plants were at least 30 days old. Seedling tests in the glasshouse cannot, therefore, be used in screening for resistance to *R. maidis*, and most selection tests have been carried out in the field. Painter and Pathak (1962) found that 70 per cent of plants of susceptible maize hybrids became infested with *R. maidis* in field tests, compared with less than 5 per cent of plants in a resistant hybrid, Kansas 1859; this hybrid was approved for release to growers in Kansas in 1950, and this and subsequently released corn varieties have made a significant contribution to the control of the corn leaf aphid in the USA.

The mechanisms involved in the resistance of hybrids such as K1859 have not been conclusively identified, but non-preference and antibiosis are probably involved. Maize lines with high concentrations of DIMBOA (see page 349) in the leaves are usually much less damaged by *R. maidis* than are those with low DIMBOA content (Long *et al.*, 1976; 1977). DIMBOA is known to be distasteful and poisonous to many insect species and may, therefore, be responsible for the antibiosis observed.

There is an interaction between *R. maidis* biotypes and certain resistant lines of maize. For example, the fecundity of *R. maidis* was twice as great on a susceptible hybrid (K2234) as on a resistant hybrid (K1859) with three biotypes, but K1859 was more susceptible than K2234 to a fourth biotype (Painter and Pathak, 1962). It has been suggested that the field resistance of K1859 in Kansas is attributable to the fact that the KS-3 biotype of *R. maidis* has been very rare in that part of the USA. However, there is no evidence that the KS-3 biotype has become adapted to K1859 and it is, therefore, probably not a true resistance-breaking race. However, these results show that resistant varieties are not necessarily resistant to all populations of *R. maidis*; selection and testing for resistance should, therefore, involve the use of aphid populations collected from as many different sites as possible.

Very little seems to be known about the inheritance of resistance to *R. maidis* in maize, but resistance appears to be complex and controlled by many genes, with varying degrees of dominance and additivity (Pathak and Saxena, 1976). However, Chang and Brewbaker (1975) reported from Hawaii that resistance is conditioned by a single recessive gene linked to the locus on chromosome 10 of gene *Rp*₁, which controls resistance to maize rust caused by *Puccinia sorghi* (see page 115). These conflicting reports can be attributed either to the extreme lability of the expression of resistance to the corn leaf aphid (Singh and Painter, 1964) or to the presence of different aphid biotypes in Hawaii and on the USA mainland.

More information is required about the genetics and mechanisms of resistance to *R. maidis* under controlled conditions, so that selection and testing procedures

can be improved and standardized. It would be particularly interesting to understand why maize seedlings are virtually immune to *R. maidis* when older plants are generally so susceptible. A better understanding of this seedling resistance might enable much higher levels of aphid resistance to be achieved in older plants.

BIRD PESTS

The yield of maize is often seriously reduced by birds feeding on the developing grain. Attempts have been made in the USA to breed maize varieties resistant to American species of 'blackbirds'. These include the red-winged blackbird, *Agelaius phoeniceus*, and are not closely related to the European blackbird, *Turdus merula*. Resistant varieties could be used to augment other means of reducing bird damage, such as chemical repellents and scare devices. Considerable progress has been made in studies of bird resistance, for example at the Delaware Field Station of the US Fish and Wildlife Service by Dr J.T. Linehan and his colleagues.

Several different methods have been used at the Delaware Field Station to compare the resistance to birds of different maize varieties. In some tests, ears of different varieties have been placed on a rack in a cage containing blackbirds so that the extent of feeding damage on the ears can be compared. The size and shape of the ears and kernels, the thickness of the husk and the sugar content of the kernels are all factors which can contribute to resistance or susceptibility. Some maize varieties have weak shanks which cause the ears to hang down at an early stage of the ripening process, so that bird damage is largely avoided. The rate of maturity can also be important in avoiding bird damage, because the grain of slow-maturing varieties will be at a susceptible stage for a longer period than grain of fast-maturing varieties.

Several bird-resistant maize varieties have been bred in the USA and some have performed well in yield trials (Linehan, 1977). There is, therefore, considerable potential for the breeding of such varieties in all the maize-growing areas where bird damage is a problem. In view of the very extensive damage by birds, this work should have the highest priority.

Alfalfa (Lucerne)

Among the most severe pests of alfalfa are two species of aphids, *Acyrtosiphum* (*Macrosiphum*) *pisum*, the pea aphid, and *Therioaphis maculata* (*Therioaphis trifolii* f. *maculata*), the spotted alfalfa aphid. Both aphids can cause serious yield losses in alfalfa crops as a result of direct feeding damage but, in addition, they can act as vectors of several virus diseases; *A. pisum* is particularly important in this respect. Resistant varieties are an important method of controlling both pests.

SPOTTED ALFALFA APHID

The spotted alfalfa aphid (*Therioaphis maculata*) thrives best in a warm, dry climate and is therefore a particularly serious pest of alfalfa in parts of the Mediterranean region, the Middle East and in the south-western parts of the USA. *T. maculata* was first observed to be causing damage in the USA in 1954,

soon after its accidental introduction. The biology of this pest and the possibilities of breeding for resistance have been extensively studied in Kansas (Peters and Painter, 1958; Harvey *et al.*, 1960).

Harpaz (1955) working in Israel, noticed that some alfalfa varieties supported lower numbers of *T. maculata* than others following natural infestation in the field. In the USA it was observed that the variety Lahontan and three of its five parental clones were resistant to the spotted alfalfa aphid (Howe and Smith, 1957) Further work showed that it is possible to select individual resistant plants from most alfalfa varieties.

The development of the variety Cody, which was bred in Kansas for resistance to spotted alfalfa aphid, has been fully described (Harvey *et al.*, 1960). Cody was derived from Buffalo which has good resistance to bacterial wilt and is adapted to Kansas conditions. Seedlings of Buffalo were grown in seed boxes in a glasshouse and were heavily infested with spotted alfalfa aphids; susceptible plants were usually killed within a month and surviving seedlings were potted-on individually and allowed to recover. These were eventually re-infested, and further selections were made. An important criterion of resistance was the percentage of infested seedlings that survived heavy infestation with *T. maculata* (Hackerott *et al.*, 1958). In other tests, aphids were caged on leaves of individual plants or on detached leaves (Thomas, Sorensen and Painter, 1966). In Cody, which was produced from 22 of the most promising plants in these tests, 82 per cent of infested seedlings survived, compared with only 65 per cent in Lahontan. Other varieties with good resistance to the spotted alfalfa aphid have been developed, for example Moapa in California (Hanson, 1961) and Zia in New Mexico (Wilson, Melton and Watson, 1959). Re-selection within existing varieties has continued with the objective of producing varieties with even higher levels of resistance.

Resistant varieties have been successfully bred without much knowledge of the underlying resistance mechanisms or a detailed understanding of the genetics of resistance. It seems, however, that resistance is complex and consists of several components, including resistance to settling (non-preference), resistance to aphid multiplication (antibiosis) and tolerance. Although aphids settle and start to feed as quickly on resistant as on susceptible plants, they become restless on resistant plants after about an hour, and eventually die or leave the plant (McMurtry and Stanford, 1960). The stylet tips of the aphids' mouthparts enter the phloem less frequently in resistant plants, and it has been suggested that aphids die on resistant plants because of starvation resulting from an inability to imbibe sufficient sap from tissues other than the phloem. Kishaba and Manglitz (1965) also found that *T. maculata* showed no initial preference for susceptible plants but that they migrated within a few hours from resistant to susceptible plants. Aphids that were confined to resistant plants died as quickly as those that had been starved.

Several distinct biotypes of *T. maculata* have been identified (Nielson *et al.*, 1971), and there is a gene-for-gene relationship between certain of these biotypes and some plants of the variety Hayden, which express corresponding genes for resistance to *T. maculata* (Nielson and Don, 1974a). However, this gene-for-gene relationship does not operate between plants of Lahontan and *T. maculata* biotypes. The significance of these interrelationships between aphid biotypes and some resistant varieties is uncertain, but no major breakdowns of resistance seem to have occurred in the field. However, four new biotypes of *T. maculata*, which are each able to attack a different set of alfalfa varieties, have recently

been identified in California (Stanford, 1977). Fortunately, Caliverde 65, which has descended from a Lahontan clone, has expressed resistance to all known biotypes of *T. maculata*.

Nielson and Don (1974b) studied the probing behaviour of different *T. maculata* biotypes on susceptible and resistant alfalfas, and concluded that resistance is based on chemical interactions between aphid and host plant, possibly involving phytoalexin-like or phenol-phenolase compounds. Kindler and Staples (1969) suggested that resistant plants may contain a repellent, or may be deficient in some nutrients that are essential for the survival of *T. maculata*. Horber *et al.* (1974) found that Lahontan contains high concentrations of saponins, which have a strong antibiotic activity against aphids and many other insects.

Only one cycle of selection for resistance in a glasshouse test was necessary to achieve a significant increase in resistance to the spotted alfalfa aphid (Harvey *et al.*, 1960). This situation, combined with the fact that alfalfa can be vegetatively reproduced, facilitated the testing and multiplication of resistant clones and made it possible for Cody to be released within five years of the first report of damage by the spotted alfalfa aphid in the USA. This was a remarkable achievement, and an important landmark in the history of pest control in plants.

PEA APHID

The pea aphid (*Acyrtosiphon (Macrosiphum) pisum*) has a very wide distribution and can colonize many species of the family *Papilionaceae* of the Leguminosae. Unlike the spotted alfalfa aphid, which can kill alfalfa plants of all ages, the pea aphid normally kills the plants in the seedling stage only. Although yield losses of more than five per cent because of direct feeding damage by *A. pisum* are unusual (Carnahan *et al.*, 1963), the importance of this aphid as a vector of several damaging virus diseases has made resistance to the pea aphid a major breeding objective, particularly in the USA.

Resistance to the pea aphid in alfalfa was first reported in 1934 and several varieties have since been shown to express some resistance. Hackerott *et al.*, (1963) devised methods of screening for resistance to *A. pisum* in the glasshouse. Large numbers of *A. pisum* were shaken over the plants to be tested, and the plants that grew vigorously and on which there were few aphids, were selected. The results of such glasshouse tests were highly correlated with those of field trials (Harvey, Hackerott and Sorensen, 1972). Similar techniques have been used in selecting for resistance to a related aphid pest, *Acyrtosiphon kondoi* (Nielson, Lehman and Kodet, 1976). Derivatives of Flemish and Turkistan-type alfalfas have been consistently resistant to *A. pisum*, although most varieties that have been examined have contained a proportion of resistant plants.

Very little is known about the nature of genetics of resistance to *A. pisum* in alfalfa. In Ladak, which has been an important source of resistance, resistance to aphid multiplication (antibiosis) and tolerance seem to be involved. Ortman *et al.* (1960) found that resistant plants often had only one-tenth as many aphids on them as susceptible plants. The multiplication rate of *A. pisum* is slower, and aphid mortality is higher, on resistant than on susceptible plants (Carnahan *et al.*, 1963). Resistance to the pea aphid in alfalfa is usually associated with a

high concentration of saponins in the tissues of the host plant (Horber *et al.*, 1974; Pedersen, Sorensen and Anderson, 1975). Saponins also reduce the growth rate and survival of the potato leafhopper and the white grub (*Melolontha vulgaris*) on alfalfa (Pathak and Saxena, 1976), but are not usually associated with resistance to several other pests, including *Therioaphis maculata*, *Meloidogyne* spp. and *Ditylenchus dipsaci* (Pedersen *et al.*, 1976). Although saponin concentration is probably an important factor in resistance to *A. pisum*, other factors are also involved. For example, resistance to the pea aphid is associated with a low protein : sugar ratio in the tissues of alfalfa plants (Wegorek and Krzmańska, 1973). According to Jones, Briggs and Blanchard (1950), resistance to *A. pisum* is controlled by a dominant gene and a closely linked gene, but further work to confirm and extend these results is needed.

Several different biotypes of *A. pisum* have been recognized. Clones of *A. pisum* differ in size and fecundity (Harrington, 1943) and in host range and host reaction (Cartier, 1963). Although it has been shown that some clones of alfalfa are more resistant to some biotypes of *A. pisum* than others (Cartier *et al.*, 1965) there is no suggestion that biotypes are a serious problem in the field at present.

Although plants that are resistant to *A. pisum* are not always resistant to the spotted alfalfa aphid (*T. maculata*), several alfalfa varieties with resistance to both aphid species have been developed in the USA (e.g. Painter *et al.*, 1965; Hunt *et al.*, 1971; Kawaguchi and Beard, 1974; Peadar *et al.*, 1976). There is clearly considerable scope for selecting within existing resistant cultivars for even higher levels of combined resistance to these aphids (Kindler and Schalk, 1975). This is an important objective because a prolonged infestation of either *A. pisum* or *T. maculata* can destroy even established plants of susceptible varieties in the field.

Potatoes

POTATO (GOLDEN) CYST NEMATODES

Potato cyst nematodes are now considered to be members of the genus *Globodera* (Mulvry and Stone, 1976), although until very recently they were included in the genus *Heterodera*. *Globodera* (*Heterodera*) *rostochiensis* and *Globodera pallida* are very widespread pests of potatoes, particularly in Europe where they can cause considerable loss of yield. They are also very common in Central and South America but have a rather limited distribution in North America (Stone, Thompson and Hopper, 1977). Control by crop rotation can be very effective and substantial increases in yield have been achieved by avoiding continuous cropping of potatoes. Until recently, crop rotation and seed certification have been the main control measures, but many areas now have such large infestations that it would be necessary to have very long rotations to achieve satisfactory control. For this reason, and to achieve a better control of potato cyst nematodes, considerable efforts have been made to breed resistant varieties.

Ellenby (1954) screened many *Solanum* spp. of the Commonwealth Potato Collection for resistance to *G. rostochiensis* by growing plants in nematode-infested soil; he considered plants to be resistant if only a few cysts developed on the roots. Using this criterion of resistance, he found that *S. vernei* and a

few clones of *Andigena* potatoes (i.e. *S. tuberosum* s.sp. *andigena*) were highly resistant. Work by Mai and Peterson (1952) and Ross (1969) showed that several other species of *Solanum*, including *S. oplocense* and *S. spegazzinii* are also resistant, as are *S. famatinae*, *S. canasense* and *S. ceptophes* (Vavilova and Zhitlova, 1976). Howard, Cole and Fuller (1970) consider that sources of resistance in *Andigena* potatoes are particularly important, presumably because, being cultivated potatoes and similar to the ancestors of *Tuberosum* potatoes, they do not have as many undesirable features as wild *Solanum* species. However, the few resistant *Andigena* clones are resistant only to certain pathotypes, whereas some accessions of *S. vernei* have general resistance to all known pathotypes (Olsen, 1969); for this reason, hybrids have been produced between *S. vernei* and *S. tuberosum*. Nevertheless, resistance to *Heterodera* spp. can also be race-specific in many wild species. Kameraz and Ponin (1974) tested clones of four wild species for resistance to three *Heterodera* populations from Peru and found that only one clone of *S. sanctae-rosae* was resistant to all three nematode populations.

Toxopeus and Huijsman (1953) showed that the resistance of CPC 1673, an *Andigena* clone, is controlled by a single dominant gene, H_1 . Dunnett (1957; 1961) found a population from Duddington, near Edinburgh, which could produce many cysts on clones with gene H_1 . He also found that the wild species, *S. multidissectum*, carries a second resistance gene, H_2 , which gave

Table 11.1 AN AGREED SCHEME FOR NAMING EUROPEAN PATHOTYPES OF *GLOBODERA ROSTOCHIENSIS* AND *G. PALLIDA* (AFTER EVANS AND STONE, 1977; KORT ET AL., 1977)

Traditional names	Agreed names	Sources of resistance
<i>G. rostochiensis</i>		
British A, Dutch A, Hilstrup	Ro1	<i>S. tuberosum</i> s.sp. <i>andigena</i> clone CPC 1673; <i>S. kurtzianum</i> clone 60.21.19; <i>S. vernei</i> clones 58.1642/4, 62.33.3 and 65.346/19
Dutch B, Obersteinbach	Ro2	<i>S. kurtzianum</i> clone 60.21.19; <i>S. vernei</i> clones 58.1642/4, 62.33.3 and 65.346/19
Dutch C	Ro3	<i>S. vernei</i> clones 58.1642/4, 62.33.3 and 65.346/19
Dutch F	Ro4	<i>S. vernei</i> clones 62.33.2 and 65.346/19
Harmertz	Ro5	<i>S. vernei</i> clone 65.346/9
<i>G. pallida</i>		
British B	Pa1	<i>S. vernei</i> clone 62.33.3 and <i>S. multidissectum</i> clone P 55/7
Dutch D	Pa2	<i>S. vernei</i> clone 62.33.3
British E, Dutch E, Frenswegen, Chavorney	Pa3	Some clones of <i>S. tuberosum</i> s.sp. <i>andigena</i> , including CPC 2802*

*Fuller, Howard and Stone, 1977

resistance to the Duddington population (pathotype B) but not to populations to which gene H_1 gives resistance (pathotype A). Later, many populations were found which could produce numerous cysts on potatoes having both genes H_1 and H_2 (pathotype E). Many populations were found also in the Netherlands and Germany which could produce a large number of cysts on potatoes with gene H_1 .

Largely because of the work of Guile (1970) (*see also* Jones *et al.*, 1970; Kort, 1974) it became clear that there are two distinct species of cyst nematodes. These are *Globodera rostochiensis*, the yellow potato cyst nematode (the golden nematode of the USA) which is characterized by remaining yellow for a long time while the cysts develop, and *G. pallida*, which has a protracted period during cyst development when it is creamy white. *G. rostochiensis* includes the original pathotype A, and *G. pallida* the original pathotypes B and E.

With the discovery of different pathotypes, breeders switched largely to *S. vernei* as the main source of resistance although other wild species including *S. kurtzianum* have also been used. Different systems of nomenclature have been employed for pathotypes in different countries (e.g. Øydvin, 1973) and it was only in 1977 that an agreed nomenclature for pathotypes was proposed for European populations of potato cyst nematodes (Kort *et al.*, 1977). In this system five pathotypes of *G. rostochiensis* (Ro1 to Ro5) and three of *G. pallida* (Pa1 to Pa3) are recognized (*Table 11.1*).

The distribution of pathotypes in the UK has been studied by Brown (1970) who showed that pathotype A of *G. rostochiensis* (now known as Ro1) is the most common pathotype in south-eastern England. Conversely, most populations of cyst nematodes in the East Midlands are pathotype E of *G. pallida* (now known as Pa3). In other areas of England and Wales there seem to be no clear-cut differences in distribution of the two species. This suggests that the differing distribution of *G. rostochiensis* and *G. pallida* is not attributable to the growing of resistant varieties in certain areas, and that resistant varieties have probably played little part in determining the present distribution of pathotypes in the UK (Saynor, 1975).

Many varieties with the H_1 gene for resistance to *G. rostochiensis* have been produced in northern Europe and some, for example Maris Piper in the UK, have become widely grown. More than one-third of the mid-early varieties and one-fifth of the late varieties grown in West Germany in 1974 were resistant to pathotype A of *G. rostochiensis*; no nematode-resistant varieties had been grown there before 1956 (Höppner, 1976). Such varieties are proving for several reasons to be very useful in areas where pathotype A populations are common. Resistant plants stimulate eggs to hatch and, although the larvae can invade resistant plants, giant cells are not commonly produced, and damage to the roots of resistant plants is correspondingly small (Howard, 1970).

Eggs of *G. rostochiensis* hatch near the roots of resistant plants, and larvae penetrate the root tissues, but cysts (mature females containing eggs) are not produced on resistant plants because the larvae either die or are predominantly male (Trudgill and Parrott, 1969). Because very few adult females develop on roots of resistant plants, the number of eggs produced on them is extremely small. It has been shown that growing a non-host crop for one year can deplete the number of *G. rostochiensis* eggs in the soil by about 25 per cent but one crop of a resistant potato can reduce the number of eggs by between 50 and 70 per cent.

Jones (1974) has suggested that there is a gene-for-gene relationship between potato cyst nematodes and potatoes which carry major resistance genes. He also postulated that all pathotypes of these nematodes can induce the formation of syncytial transfer cells in all potato genotypes but only do so when there is no incompatible interaction between the saliva of individual nematode pathotypes and the host plant tissues.

Although the main emphasis has been on resistance to cyst formation, differences in tolerance between potato varieties have been reported by Huijsman, Klinkenberg and Den Ouden (1969). They found that in the Netherlands the varieties Multa and Panther, which are susceptible to *G. rostochiensis* and *G. pallida*, gave good yields in soils heavily infested with cyst nematodes. This was thought to be caused by vigorous regeneration of affected roots and resistance to secondary root rotting organisms. The growth of Maris Piper (resistant to pathotype A of *G. rostochiensis*) is less retarded in heavily infested soils than is that of susceptible varieties (Evans, Parkinson and Trudgill, 1975). Resistant varieties can also withstand water stress better than susceptible varieties.

There is an urgent need for varieties that are resistant to *G. pallida*. Howard, Cole and Fuller (1970) suggested that some Andigena clones carry a gene H_3 which controls resistance to several *G. pallida* populations. This resistance is being used in breeding programmes, although recent work suggests that several genes rather than one are involved. Landeo, Rowe and Mayer de Scurrah (1976) tested several Andigena clones for resistance to two populations of *G. pallida* collected from different parts of Peru. Three of the clones examined were resistant to one population, this resistance being controlled by a single, dominant gene. Three other clones were resistant to the second population but their resistance was controlled polygenically. Two further Andigena clones expressed partial resistance to both populations.

Ross (1969) in Germany, and Kort, Jaspers and Dijkstra (1972) in the Netherlands, have tried to obtain resistance to both *G. rostochiensis* and *G. pallida* from wild *Solanum* species, particularly *S. vernei*, a diploid species from Argentina. *S. vernei* is claimed to be resistant to all known pathotypes of *G. rostochiensis* and *G. pallida*, and its resistance may therefore be non-race-specific. Although this form of resistance might be of more general use in agriculture than resistance derived from Andigena, its inheritance is complex, probably involving several major and minor resistance genes (Ross, 1969). These genes can apparently be separated in breeding programmes so that *S. vernei* derivatives can be used to identify different pathotypes (Kort *et al.*, 1977). Fuller and Howard (1974) consider that the three most important sources of resistance to *G. pallida* are *S. vernei*, *S. multidissectum* (which carries gene H_2) and Andigena stocks CPC 2775 and 2802 (which probably carry gene H_3).

Preliminary screening tests for resistance to *Globodera* spp. have usually been carried out in the glasshouse. In such tests, plants are grown in infested soil in pots and the number of cysts on the outside of each root ball is counted. Those which appear resistant in this 'root-ball' test are then re-tested more thoroughly using a soil-flotation method by which cyst numbers can be determined accurately (Howard and Fuller, 1975). Huijsman (1975) advocates a more refined technique of screening in which individual plants in pots are infected with a known number of *Globodera* cysts, eggs or larvae of a known pathotype; cysts on the roots of each plant are then counted at the end of the season. This method gives results which agree closely with those of field tests.

Engel and Stelter (1974) have devised a method for predicting the effects of different sources of resistance to *Globodera* spp. on the populations of nematodes in the field. The number of *Globodera* larvae per 100 cm³ of soil is taken as the threshold between resistant and susceptible varieties. Low density values for a particular variety indicate that the nematode population will decrease if that variety is grown continuously.

Resistant potato varieties are providing a useful measure of control of the potato cyst nematodes, particularly when they are used in conjunction with other control measures, such as crop rotation and nematicides. The resistance that is available in current European varieties is ineffective against many populations of *G. rostochiensis* and *G. pallida*, including most pathotypes from the Andes region of South America (Evans and Stone, 1977). Varieties with more broadly based resistance are therefore urgently required.

APHIDS

Although it has been known for many years that aphids (*Myzus persicae* and *Macrosiphum euphorbiae*) carry several important potato viruses (see page 231), only recently have attempts been made to breed for resistance to aphids. Adams (1946) first reported differences between species of *Solanum* in resistance to *M. persicae*, the most important virus vector in potatoes. Serghiou (1968) found a small degree of resistance to *M. persicae* in *Solanum medians*, *S. bukasovii*, *S. simplicifolium*, *S. famatinae*, *S. microdontum*, *S. stoloniferum* and *S. sanctae-rosae* and a higher level of resistance in *S. brachistotrichum*. Radcliffe and Lauer (1968; 1970) reported that several wild *Solanum* species show a high level of resistance to *M. persicae* and to *Macrosiphum euphorbiae*. *Solanum bulbocastanum*, *S. michoacanum* and *S. steriophyllidium* are resistant to *M. persicae*, and *S. bulbocastaniensis*, *S. hjertingii*, *S. polytrichon* and *S. stoloniferum* are resistant to *M. euphorbiae*. Several other wild species including *S. canasense* (Tarn and Adams, 1973) and some tetraploid *Solanums* (Tingey and Plaisted, 1976) are good sources of resistance. Varietal differences in aphid resistance are also present in certain *S. tuberosum* varieties (Canada Department of Agriculture, 1972), but are probably too small to have a major impact on the reduction of direct aphid feeding damage or on virus transmission by aphids.

In studies of aphid resistance in *Solanum chacoense*, *S. stoloniferum* and *S. demissum*, Gibson (1971a) showed that *Macrosiphum euphorbiae* had a strong preference for *S. chacoense* and least preference for *S. stoloniferum*. These results confirmed the conclusions of Radcliffe and Lauer (1970) that resistance to *M. persicae* and *M. euphorbiae* probably involve different mechanisms. However, it might be difficult to employ the aphid resistance in these species because selecting for resistance to one aphid species might result in greater susceptibility to the other.

Resistance to aphids can be assessed in the field, or on intact plants or excised leaflets in the glasshouse or laboratory. There is a good correlation between the results of tests with artificially infested leaflets and naturally infested field plants (Sams, Lauer and Radcliffe, 1975; 1976). In excised-leaf experiments with progenies of 57 tuber-bearing *Solanum* spp., Sams *et al.* (1975) found that aphid

resistance was partially dominant and heritability estimates ranged from 50 to 60 per cent in both diploid and tetraploid potatoes. Excised leaflet tests seem to be very appropriate for preliminary screening for resistance to aphids, but clones that are selected for resistance should then be re-tested in replicated field trials.

Some kinds of resistance to *M. persicae* and *M. euphorbiae* in *Solanum polyadenium*, *S. tarijense* and *S. berthaultii* are associated with the presence of glandular hairs on the leaves and stems (Gibson, 1971b). When aphids rupture the cell walls of these glandular hairs during movement over the leaf surface, a sticky liquid exudes from the hair cells and contaminates their limbs. This effectively immobilizes many of the aphids to such an extent that they starve to death. The older leaves of these three species were found to be very susceptible to both types of aphid, presumably because there were fewer hairs on these leaves and there was less exudation from them. The abundance of hairs in different potato clones is therefore a good indication of their resistance to aphids (Gibson, 1976a). This form of aphid resistance would probably give a very good control of aphids, particularly in combination with non-preference resistance. This would encourage restlessness of aphids on resistant plants and they would become immobilized by the gummy exudates even more quickly. The resistance associated with these hairs is likely to be equally effective against all aphid species and biotypes.

If resistance to aphids can be transferred successfully from wild *Solanum* species to cultivated varieties, and if the resistance is determined by a few genes only, there could be excellent prospects for controlling both aphids and aphid-transmitted viruses by resistant varieties. A relatively simple means of inheritance is essential in such cases because breeding from a wild species must include several backcrosses to susceptible cultivated varieties.

CONCLUSIONS

Very good progress has been made in breeding potato varieties with resistance to *Globodera rostochiensis*, and varieties with resistance to *G. pallida* should be available soon. Much of the resistance is highly race-specific, however, and resistance-breaking pathotypes are a serious danger. Higher levels of more durable resistance might be achieved by varieties in which several different kinds of monogenically and polygenically inherited resistance mechanisms are combined, as suggested by Hermesen (1975). A combination of resistance to cyst development and tolerance to nematode damage might be particularly useful, because resistant varieties would be highly productive even in heavily infested soils and would reduce nematode populations in the soil, to the benefit of succeeding crops. On the other hand, integrated control using both nematicide treatment and resistant varieties is now being used successfully. This is economic because many of the new nematicides are relatively cheap and the potato crop often has a high cash value.

Although no varieties have been produced with a significant level of resistance to aphids, resistant varieties could make a very significant contribution to the control of aphid-transmitted viruses, such as leaf roll and potato virus Y.

Clover

STEM NEMATODES

Ditylenchus dipsaci is an important pest of several crops, including red clover, white clover and oats. It is widely distributed in northern Europe, Canada and the northern parts of the USA, where it can seriously reduce the yield of red clover (*Trifolium pratense*). Patches of stunted plants are commonly seen in crops of susceptible varieties grown on heavily infested soils, and the aerial parts of infested plants become distorted. Several morphologically indistinguishable biotypes (pathotypes) of the stem nematode, with distinct host ranges, have been identified; for example, red and white clovers are apparently attacked by different races of *D. dipsaci*. Resistant varieties of red and white clover are already helping to control the stem nematode in several countries. Such varieties yield more green matter, and become established more quickly when grown in infested soils, than do susceptible clovers (Williams and Barclay, 1972).

Pioneer work on resistance to *D. dipsaci* in clover was carried out in Sweden by Bingefors (1951) who reported that the varieties Merkur and Resistenka, which had been derived from plants selected for resistance in heavily infested soil, were resistant. Later work by Bingefors (1957) showed that Merkur was resistant to all populations of *D. dipsaci* against which it had been tested, and gave a good control of the pest in several different parts of Sweden. The green-matter yield of Merkur was reduced by only 20.4 per cent compared with a 39.4 per cent reduction in susceptible varieties in heavily infested soils in Germany; the yield of another resistant variety, Bora, was not significantly reduced on infested soil (Spanakakis, 1973a). The proportion of infested plants in these experiments were 37.7, 45.1 and 78.5 per cent in Bora, Merkur and the susceptible controls respectively, suggesting that non-preference or some other form of pest escape is responsible for the resistance. Hungaropoly is tolerant to *D. dipsaci* because, although this variety becomes infested, it is not severely damaged as a result.

Several techniques for testing the susceptibility of red clover seedlings to stem nematode in the laboratory have been developed (e.g. Bingefors, 1957; Toynbee-Clark and Bond, 1970; Dijkstra and Koster, 1973). In one method, seedlings are grown between layers of moist filter paper and one drop of a suspension of *D. dipsaci* larvae is applied to each seedling; after two or three weeks, the seedlings are scored for symptoms. Toynbee-Clarke and Bond (1970) compared the severity of symptoms on different plants using the following scoring system:- (0) no swelling; (1) no swelling but with some necrosis; (2) stunted, no swelling; (3) slight swelling of hypocotyl; (4) greatly swollen petiole; (5) greatly swollen hypocotyl. Seedlings from classes 0 and 1 were selected for further tests and for use in breeding. Nüesch (1971) used a similar scoring system but recognized only three classes:- (1) necrosis but no swelling of the hypocotyl; (2) swollen hypocotyls; (3) no symptoms. These kinds of seedling tests have effectively distinguished between varieties and breeding lines that are known to differ in resistance to stem nematode in the field. For example, the red clover variety Essex Broad Red, which is very susceptible to *D. dipsaci* in the field, had many more grossly swollen seedlings in laboratory tests than Dorset Marl, which has good field resistance to this nematode. Clover varieties or lines in which less than 40 per cent of inoculated plants become badly

infested with *D. dipsaci* in such experiments, have usually had an adequate level of resistance to stem eelworm in the field (Dijkstra and Koster, 1973). A single generation of selection using these methods has often resulted in a significant increase in resistance. Similar methods have been used to select resistant lines of *T. repens*. Williams (1972) tested many lines of white clover for resistance to *D. dipsaci* in the laboratory but found that only six (all Ladino clovers) were resistant. These have been hybridized with locally adapted clovers for use in New Zealand.

Armstrong, Pinkerton and Jensen (1977) grew seedlings of 682 red clover accessions in flats (seed trays) of damp soil in the glasshouse. The soil was watered with a suspension of nematodes (42000 to 52000 nematodes per litre) one day after the seed was sown. Accessions in which more than 40 per cent of seedlings became infested were rejected, the remainder being re-inoculated in similar tests. Major differences in susceptibility were observed, and four accessions (Great Britain 306–193, Sweden 304–783, Denmark 234–703 and South Africa 300–153) were particularly resistant.

Nematode-resistant clover varieties have been developed in several countries. For example, a resistant tetraploid red clover, Maris Leda, has been marketed in the UK; this doublecut variety has a high level of resistance to *D. dipsaci*, derived partly from a Swedish variety Silo (itself a derivative of Merkur) and partly from Pearse's, a nematode-resistant stock of Dorset Marl (Bond, 1960). A medium-late diploid variety, Britta, has been developed in Sweden from a cross between a Danish variety, Daeno, and Resistanta; more than 50 per cent of plants of Britta are resistant to *D. dipsaci* (Jönsson, 1974).

Very little is known about the mechanisms of resistance to *D. dipsaci*. In resistant Swedish varieties, the development and reproduction of the nematode is impaired in resistant plants (Bingefors, 1951; 1957). This implies that antibiosis is involved in resistance, in addition to non-preference and tolerance. A lack of nematode-induced swelling of the hypocotyls of infested plants, which has been used as a manifestation of resistance by many plant breeders, is presumably an indication of tolerance.

The inheritance of resistance to *D. dipsaci* seems to be complex, which is to be expected because of the different resistance mechanisms involved. Bingefors (1956) found that progenies of resistant $\times$ resistant parents are generally resistant and those of crosses between susceptible parents are usually susceptible; most progenies of resistant $\times$ susceptible crosses show an intermediate level of resistance, but some are unexpectedly resistant and others are very susceptible. Although other workers have also found that the resistance of Swedish varieties is not simply inherited, Nordenskiöld (1971) reported that resistance in some Swedish varieties is controlled by two dominant genes, one of which is closely linked to the *S* (self-incompatibility) locus; selection for nematode-resistant individuals in a clover population usually results in an increased frequency of particular *S* alleles. Selection for increased frequency of certain *S* alleles might, therefore, be a means of selecting for resistance to *D. dipsaci*. Resistance to *D. dipsaci* in Pearse's stock of broad red clover is apparently also inherited as a dominant character (Dr D.A. Bond, unpublished data). These conflicting reports concerning the genetics of stem nematode resistance in clover have probably arisen because different methods of assessing resistance have been used by different workers, and because some components of resistance are controlled by dominant major genes and others by polygenes.

Spanakakis (1973b) found that there was a significant interaction between four red clover varieties and seven populations of *D. dipsaci* collected from different parts of Germany; however, no races or biotypes of *D. dipsaci* were shown to have become adapted to specific resistant varieties. Merkur has been resistant to all the populations of *D. dipsaci* against which it has been tested in Sweden, and resistance-breaking biotypes are unlikely to be a major problem. Wallace (1963) has pointed out that resistant varieties of several crop species have given good control of stem nematodes for many years, largely because of the absence of resistance-breaking biotypes.

Tobacco

ROOT-KNOT NEMATODE

Root-knot nematodes (*Meloidogyne* spp.) are the most widespread and damaging nematode pests of tobacco. Their effects are particularly serious in the south-eastern parts of the USA, Rhodesia and Australia, and are most common on light, sandy soils. Infested plants have characteristic galls on their roots, their growth is stunted and there is wilting of the leaves. The yield and quality of the leaves of affected plants can be seriously reduced. The most important species in the USA is *M. incognita* var. *acrita*, but in Queensland, Rhodesia and East Africa, *M. javanica* is the most prevalent species (Akehurst, 1968).

Resistance to *Meloidogyne* has been known for a long time but its incorporation into satisfactory commercial varieties has been difficult (Clayton *et al.*, 1958). Moore *et al.* (1962) developed a variety, NC95, which is resistant to *M. incognita*, for use in North Carolina; this variety has been a major source of resistance for other breeders. *M. incognita* does not produce galls on the roots of NC95 but its yield can be seriously reduced by the nematode in heavily infested soils. Resistance to *M. incognita* from a South American tobacco strain, TI 706, which is controlled by a single dominant gene and several modifying genes, has also been extensively used by plant breeders. Unfortunately, resistance has usually been closely associated with undesirable characteristics, including small and narrow leaves, and this has greatly hindered the incorporation of resistance to root-knot nematodes into commercially acceptable varieties. Graham (1961) developed a resistant breeding line, PD611, with resistance derived from TI 706, which was crossed with an allopolyploid from *Nicotiana sylvestris* × *N. tomentosiformis*; this has been released to breeders as a promising source of resistance to *M. incognita*. Varieties that have been bred from PD611 and related sources of resistance have given some control of *M. incognita* in the USA but are susceptible to *M. javanica* or *M. arenaria* (Graham, 1961). These two latter species of *Meloidogyne* are rare in the USA at present but may become more common if there is an increased popularity of varieties with resistance only to *M. incognita*.

Resistance to *M. javanica* has been found in some Rhodesian tobaccos by Schweppenhauser, Raeber and Daulton (1963) but, as with *M. incognita*, resistance to *M. javanica* is often associated with undesirable characters. However, the development of satisfactory flue-cured tobaccos is being attempted in

southern Africa by producing aneuploid breeding lines of *N. tabacum* so that root-knot nematode resistance can be conveniently transferred from *N. longiflora* and *N. repandra* into commercially desirable genetic backgrounds. Resistance in *N. longiflora* is controlled by a single dominant gene when nematode populations are relatively low, but linked modifying genes affect the expression of resistance under conditions of heavy nematode infestations (Schweppenhauser, 1975a,b). *Nicotiana nudicaulis*, *N. plumbaginifolia*, *N. landsdorffii*, *N. nesophila* and the cultivated tobacco Florida 22 and line SC66 are also resistant to *M. javanica* (Muro, 1972). Stavely, Pittarelli and Burk (1973) successfully transferred resistance genes from *N. repandra* to cultivated tobaccos but these genes were progressively lost in succeeding backcross and selfed generations. Schweppenhauser (1974), who also used chromosome addition lines of tobacco to transfer resistance from wild species, encountered similar difficulties. Fortunately, some worth-while sources of resistance to *M. javanica* have recently been found in some *N. tabacum* lines (Schweppenhauser, 1975a) and these should be easier for plant breeders to exploit than those from wild *Nicotiana* species.

Nematode-resistant varieties, such as NC95, sometimes become badly infested with *M. incognita* and genetic variations in virulence towards certain varieties may be partly responsible for these attacks (Parvatha Reddy, Setty and Govindu, 1972). However, resistance-breaking biotypes of *M. incognita* have not been a problem in the field. Many tobacco varieties that are resistant to *M. incognita* have been marketed in the USA and these must have significantly reduced the amount of root-knot nematode damage. These include many 'Coker' varieties, some of which have multiple pest and disease resistance. For example, Rogers (1975) reports that Coker 354 is highly resistant to *M. incognita*, *Phytophthora parasitica* (downy mildew), *Pseudomonas solanacearum* (bacterial wilt), *Fusarium* wilt and *Alternaria longipes*.

Raspberries

APHIDS

Aphid-transmitted virus diseases are common causes of serious yield losses in raspberry crops throughout the world. The most important vector of these viruses in Europe is *Amphorophora idaei* which was, until recently, mistakenly classified as *A. rubi*; *A. rubi* is now known to infest blackberries rather than raspberries (Blackman, Eastop and Hill, 1977). A different species of *Amphorophora*, *A. agathonica*, is the main vector of raspberry viruses in North America (Kennedy, Day and Eastop, 1962), although *Aphis rubicola* can also be a serious pest. This misidentification of the aphid species concerned has led to a very confused situation in breeding for aphid resistance in raspberries (Blackman *et al.*, 1977). Briggs (1959) showed that whereas European populations of *A. idaei* (then referred to as *A. rubi*) bred on Lloyd George, American populations of *Amphorophora* did not. It is now known that these American populations were *A. agathonica* and not biotypes of *A. idaei*. American varieties of *Rubus strigosus* (e.g. Chief), on the other hand, are resistant to most populations of *A. idaei* from Europe. Breeding for resistance to aphids therefore mainly has concerned *A. idaei* in Europe and *A. agathonica* in North America.

Major genes for resistance to *A. idaei* have been found in several *R. idaeus* varieties (e.g. Baumforth A) and *R. strigosus* lines and varieties (e.g. line L 518

and Chief) and in many wild *Rubus* species (Keep, Knight and Parker, 1970). There is a specific interaction between some of the resistance genes and individual *A. idaei* biotypes, whereas other resistance genes are effective against all known populations of this aphid species. The relationships between the four main *A. idaei* biotypes that are recognized in the UK and different resistance genes are summarized in *Table 11.2*.

Table 11.2 INTERRELATIONSHIPS BETWEEN BIOTYPES OF *AMPHOROPHORA IDAEI* AND SOME RESISTANCE GENES IN *RUBUS* SPP. (AFTER KEEP *ET AL.*, 1970)

Resistance gene	Source of resistance	Resistance (R) or susceptibility (S) to four biotypes of <i>A. idaei</i>			
		1	2	3	4
	<i>Rubus idaeus</i>				
A ₁	cv. Baumforth	R	S	R	S
A ₂	cv. Chief	S	R	S	S
A ₅	cv. Chief	R	S	S	S
A ₆					
A ₇					
	<i>Rubus idaeus strigosus</i>				
A ₈	Line L 518	R	R	R	R
A ₉					
	<i>Rubus occidentalis</i>				
A ₁₀	cv. Cumberland	R	R	R	R
A ₁₁	<i>Rubus coreanus</i>	R	R	R	R

Many new sources of resistance to *A. idaei* have been identified in wild raspberries, including *R. odoratus*, *R. procerus*, *R. henryi* and *R. coreanus*; (Keep, 1972; Anisimova, Kichina and Pomazkov, 1974). However, most of these are valueless because of their small fruit and spindly canes (Keep, Knight and Parker, 1970). Nevertheless, major resistance genes occur very commonly in wild raspberries and some of these will undoubtedly prove to be very useful in breeding for resistance to aphids.

Selecting and testing for resistance to *A. idaei* has usually been carried out in the field under conditions of heavy, natural aphid infestations. However, laboratory and glasshouse screening methods have been developed (*see Figure 11.6*). For example, Keep and Knight (1967) developed a simple but very effective method of assessing the resistance of raspberry seedlings or first-year canes to *A. idaei* in the insectary. Plants were each infested with small numbers of adult aphids; they were classed as susceptible if larvae were freely produced, and resistant if the aphids walked off. This kind of test is particularly suitable for detecting varietal differences in non-preference forms of resistance.

Several raspberry varieties with resistance to *A. idaei* have been marketed. Malling Exploit, Malling Landmark, Wädenswil Red, September and Gradina are immune or highly resistant to *A. idaei* (Anisimova *et al.*, 1974; Živanović, 1974). Another variety, Malling Delight, which includes Malling Promise, Lloyd George and Baumforth A in its parentage, carries the gene A₁, which confers resistance to 'strains' 1 and 3 of *A. idaei* (Keep and Parker, 1974). Leo, which was developed from a cross between an aphid-resistant species, *R. occidentalis*,

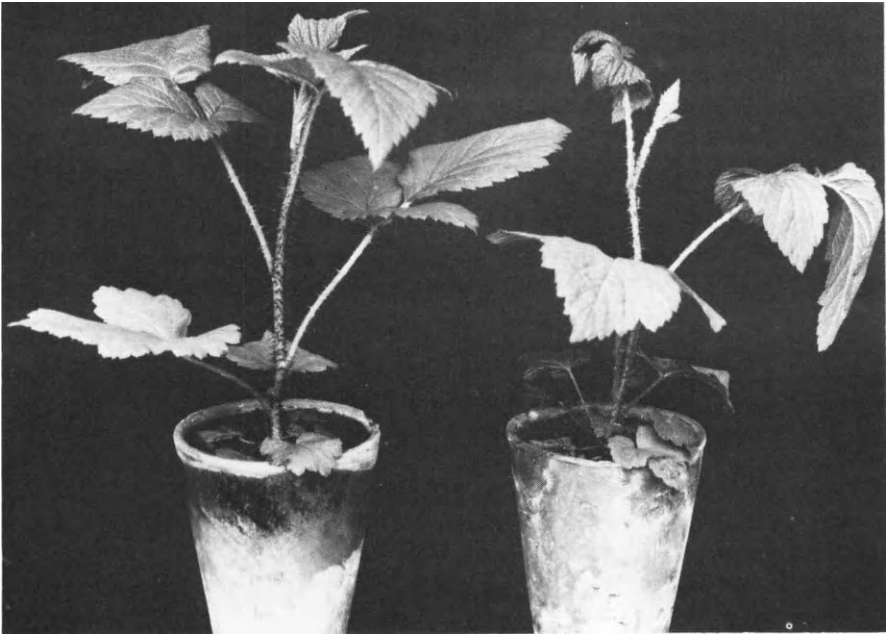


Figure 11.6 Raspberry seedlings carrying genes (A_1a_1) for resistance to the raspberry aphid are not colonized or damaged by most biotypes of *Amphorophora idaei* (left). Conversely aphids multiply rapidly on susceptible seedlings (right) and can distort the leaves. (By courtesy of East Malling Research Station).

and a cultivated red raspberry, carries the A_{10} gene for resistance to all four of the main *A. idaei* biotypes in the UK (Keep, 1975). Glen Clova, with partial resistance to *A. idaei* controlled by several minor genes, has also been marketed in the UK (Jones, 1976).

Aphid-resistant varieties have had a much lower incidence of aphid-transmitted virus diseases than susceptible varieties in field experiments (Jones, 1976). For example, two varieties (Lloyd George and Malling Jewel) that are susceptible to *A. idaei* became heavily infested with aphids and badly affected by virus diseases, whereas the aphid-resistant varieties Glen Clova, Norfolk Giant and Malling Orion had few aphids and little virus infection. These results show that resistance to the aphid vectors can play an important part in controlling virus diseases in the field.

In large-fruited raspberries there is a correlation between resistance to *A. idaei* and suitability for deep-freezing of the fruit; most susceptible varieties lose more juice and have poorer quality than aphid-resistant varieties after freezing (Bauer, 1973). To offset this unexpected advantage of aphid resistance, however, resistant varieties usually have paler fruits and juice than susceptible varieties.

Unexpectedly high numbers of *A. idaei* have sometimes been observed on certain resistant varieties in Scotland (Woodford and Dickson, 1975). However, there is no firm evidence that these attacks are caused by resistance-breaking biotypes of *A. idaei*. No new resistance-breaking biotypes of *A. idaei* have been isolated in the UK despite increasing selection pressure in their favour, and the use of major resistance genes may provide a lasting control of raspberry aphids

in Europe (Keep *et al.*, 1970; Knight and Alston, 1974). Lloyd George shows a high level of aphid resistance in North America but not in Europe, because it is resistant to *A. agathonica* but not to *A. idaei*. In the 1930s, Schwartze and Huber (1939) tested several raspberry varieties that were commonly grown in the USA, for resistance to the *Rubus* aphid. They found that Indian Summer, Lloyd George, Pyne's Imperial and Pyne's Royal were highly resistant to what is now known to be *A. agathonica*; aphids failed to reproduce on plants of these varieties. Some other varieties, including Antwerp, Herbert and Newburgh, showed a lower level of aphid resistance, and many of those tested were very susceptible. The resistance in Lloyd George is a partially dominant character (Schwartze and Huber, 1939; Daubeny, 1966). The resistance of many varieties, including Lloyd George, Pyne's Royal and Canby, has continued to give a good control of *A. agathonica* in North America for many years (Daubeny, 1972; Kennedy, Schaefer and Ourecky, 1973); there is no conclusive evidence that aphid populations have become adapted to any of these varieties in spite of the fact that they have been planted over large areas. Nevertheless, the resistance of some varieties has been less effective in some parts of North America than others, which suggests that resistance-breaking populations may exist (Converse *et al.*, 1971). Alternatively, populations of *A. agathonica* may be unusually high on all varieties in certain areas; or *A. idaei*, which was introduced accidentally into North America from Europe in the 1960s (Blackman *et al.*, 1977), may have become established in certain parts of the USA and Canada, and most American varieties have never been resistant to this species.

Natural aphid infestations have generally been used in the field for evaluating resistance to *A. agathonica*. However, laboratory tests have been developed which give results that agree closely with those from field experiments. For example, Kennedy and Schaefer (1974a) infested individual raspberry cuttings with a single apterous adult aphid, and the number of aphids on each cutting was recorded after three days. In field and laboratory tests, plants with very few or no aphids were considered to be resistant and were selected for use in breeding. Both non-preference and antibiosis types of resistance to *A. agathonica* have been reported. Non-preference is expressed in Canby and Malling Exploit, and antibiosis (which is identified by the slow development and premature death of aphids) is expressed in the variety Washington (Kennedy and Schaefer, 1974b). Canby also shows antibiosis, which may be related to a low concentration of sugars and nitrogenous compounds in the phloem sap in the leaves (Kennedy and Schaefer, 1975).

CONCLUSIONS

Good sources of resistance to raspberry aphids are available in cultivated raspberries and wild *Rubus* spp., and these have been successfully exploited in many breeding programmes. In North America, many raspberry varieties with excellent resistance to *A. agathonica* are available and this resistance has been durable, with no major problems of resistance-breaking aphid biotypes. In Europe, too, many varieties with resistance to *A. idaei* have been marketed, although many of the resistance sources used are known to be race-specific. However, the resistance has been durable in the field and the use of major resistance genes should provide a lasting control of raspberry aphids.

Aphid resistance has given a good control of aphid-transmitted raspberry viruses but this should be combined with resistance to the virus diseases themselves (see page 260) to achieve an even better disease control, as suggested by Jennings (1963). Such a combination of virus resistance and aphid resistance should be more effective than either type of resistance alone, and would provide an insurance against the development of new resistance-breaking aphid biotypes.

Brassica Crops

CABBAGE APHID

The cabbage aphid (*Brevicoryne brassicae*), which is a serious pest of *Brassica* crops almost everywhere they are grown, can multiply very rapidly under favourable conditions and then forms large, dense colonies on leaves, stems and inflorescences. Such infestations cause severe distortion of the leaves and can significantly decrease yield through direct feeding damage. *B. brassicae* is also an important vector of certain viruses, particularly cauliflower mosaic virus, cabbage black ringspot and turnip mosaic viruses. In addition, the presence of even small numbers of aphids on some horticultural *Brassica* crops can greatly reduce the marketability of the produce. Insecticides can control cabbage aphids fairly effectively in many horticultural crops, particularly Brussels sprouts, but some of the chemicals used have a high mammalian toxicity and are expensive. It is often uneconomic to control aphids by insecticides in forage *Brassicaceae* and in oil seed rape crops. For these reasons, attempts have been made to breed for resistance to aphids in some *Brassica* crop plants, particularly Brussels sprouts, rape and kale.

Extensive work on resistance to cabbage aphid in forage rape (*Brassica napus*) has been carried out in New Zealand. In 1947 a programme was started to transfer aphid resistance from two swede varieties, Sensation and Calder, to forage rape. These swede varieties are not immune to this aphid but generally support smaller aphid populations and they are damaged less by aphid attack than more susceptible varieties (Lamb, 1953). Palmer (1960) selected resistant plants from crosses between Calder swede and aphid-susceptible rape stocks and from these, Aphid Resistant was developed. This variety, which gives lower yields under aphid-free conditions than most aphid-susceptible rapes, but much higher yields when aphid infestations are severe, was released to growers in New Zealand in 1961; by 1966 it occupied more than 90 per cent of the rape acreage in that country (Palmer and Smith, 1967). A higher-yielding aphid-resistant variety, Rangi, which is more palatable to sheep than Aphid Resistant, was subsequently developed and this also became widely grown in New Zealand (Lammerink, 1968).

Although most testing and selection for resistance to the cabbage aphid has been carried out in the field, efficient methods of selecting in the glasshouse have also been devised. The leaves of aphid-susceptible rapes, but not those of resistant varieties, become severely distorted in the glasshouse after infestation with *B. brassicae*; this indicates that tolerance is an important factor in aphid-resistant rape varieties (Smith, 1968). There is a strong positive correlation between the severity of leaf distortion in the glasshouse and aphid damage in the field.

In 1967, cabbage aphids caused severe damage on four crops of aphid-resistant rape in the Canterbury area of New Zealand (Lammerink, 1968). The severely damaged crops were infested with a hitherto unrecognized biotype of *B. brassicae*, which reproduced more quickly and caused much more damage on all the rape varieties tested in a series of experiments, including Aphid Resistant and Rangi. This biotype is, therefore, not specifically adapted to any particular aphid-resistant variety and is thus not strictly resistance-breaking. However, if such vigorous biotypes were to become widespread, aphid-resistant varieties would suffer greater aphid damage than in the past, but susceptible varieties would presumably be affected to an even greater extent.

Some of the factors that are involved in the resistance of Aphid Resistant rape were studied by Dunn and Kempton (1969). Only about half as many immigrant alate (winged) cabbage aphids settled to reproduce on the resistant variety as on a susceptible rape, indicating that a strong non-preference type of resistance is expressed in Aphid Resistance. The winged aphids that settled on resistant plants reproduced more slowly and *B. brassicae* larvae took longer to mature on them than on susceptible plants. In addition, the reproductive life of apterous forms of the aphid was shortened on resistant plants, their fecundity was greatly reduced and many of their progeny died before maturity. This suggests that there is a very high level of antibiosis in Aphid Resistant rape in addition to non-preference resistance. The resistance to *B. brassicae* was effective in Aphid Resistant only before the onset of flowering, after which it became as susceptible as other varieties.

The resistance to cabbage aphids that has been derived from the swedes Calder and Sensation is, therefore, apparently based on non-preference, antibiosis and tolerance. In spite of the fact that resistant plants can still be damaged by some biotypes of *B. brassicae*, aphid-resistant rape varieties can give a very good control of cabbage aphids under most conditions (Lowe, 1969).

Plants of some breeding lines of forage kale (*Brassica oleracea* var. *acephala*) have non-waxy leaves, and these are less frequently colonized by *B. brassicae* than those with waxy leaves (Thompson, 1963). These non-waxy plants are also resistant to the cabbage white fly (*Aleuroides brassicae*). Thompson (1963) suggested that, as both these insects secrete a mealy covering of wax over their bodies, an absence of wax or of a precursor of wax in non-waxy leaves of host plants might be responsible for this resistance. However, resistance is often also associated with unusually low concentrations of sinigrin, a mustard oil glucoside, which stimulates host selection by *B. brassicae* (Wensler, 1962).

Brussels sprouts with waxy leaves also generally support larger populations of cabbage aphid than those with glossy or non-waxy leaves, but *Myzus persicae* prefers to settle on glossy leaves rather than on waxy leaves (Way and Murdie, 1965). Many natural enemies of *B. brassicae*, for example Coccinellidae and Anthorcoridae, prefer to oviposit on non-waxy leaves, and this may contribute to the aphid resistance of non-waxy plants. Although glossy, non-waxy Brussels sprouts have a potentially valuable degree of resistance to cabbage aphids, they are unusually susceptible to several other insect pests, including *Myzus persicae*, which is a more efficient vector of many viruses than is *B. brassicae*.

Dunn and Kempton (1971) identified non-preference, antibiosis and tolerance in some European Brussels sprouts varieties and inbreds. For example, several varieties, including Roodnerf, Gravendeel, Rollo, Stickema and Darkmar 21, were tolerant and suffered little damage from infestations of *B. brassicae*.

although they were often heavily infested. Conversely, Irish Elegance suffered great damage as a result of *B. brassicae* infestations, although the aphid populations on this variety were not exceptionally high. The leaves of sensitive or intolerant varieties were particularly prone to incurling of the foliage.

Dunn and Kempton (1972) selected individual plants for resistance to cabbage aphids from field plots of open-pollinated Brussels sprouts varieties, and tested their progenies for resistance to *B. brassicae*. Most progenies of selected plants were more resistant than the parent variety, and expressed strong antibiosis and non-preference to this aphid. Biotypes of *B. brassicae*, which were collected from different areas of England, differed in their ability to colonize different sprouts clones, so that individual plants were resistant or susceptible according to the particular biotype with which they had been infested. Biotypes of *B. brassicae*, which are adapted to particular resistant varieties may, therefore, become widespread and damaging if these varieties are grown extensively. For this reason, Dunn and Kempton (1972) were not optimistic about the long-term prospects of breeding for aphid resistance in Brussels sprouts. However, aphid-resistant Brussels sprouts varieties have been marketed in Europe including Marster's Special, Yates' Climax, Vanguard and Wearmouth, which are partially resistant to cabbage aphids, and Gronalto and Early Halfall which are tolerant (National Vegetable Research Station, 1972; Dunn and Wheatley, 1975).

Biotypes of *B. brassicae* that are adapted to colonize resistant rape and Brussels sprouts plants more effectively than others, seem to pose a serious threat to the long-term control of the cabbage aphid by resistant varieties. Nevertheless, many different kinds of resistance to cabbage aphids are available to the plant breeder, including non-preference, antibiosis, tolerance and enhanced suitability for colonization by natural enemies of aphids. A combination of several of these types of resistance in a single variety would probably adequately control all biotypes of *B. brassicae* for many years, although other additional methods of control might occasionally be necessary. It is not necessary to achieve a complete control of aphid pests by resistant varieties in order to obtain a satisfactory reduction in pest damage. Even small differences in heritable resistance can hamper a pest to such an extent that other forms of control, either chemical or biological, become much more effective. Resistant varieties should, therefore, become the basis of integrated control of pests such as cabbage aphid, whenever possible.

Lettuce

ROOT APHID

The root aphid (*Pemphigus bursarius*) is an important pest of summer lettuce, particularly in the UK and parts of northern Europe and the USA. Winged females of *Pemphigus bursarius* migrate from poplar (the alternate host) in the Spring to colonize the roots of summer lettuce plants. Under favourable conditions very large aphid colonies can develop on the roots of susceptible plants, which then wilt and die. It is difficult to predict when severe attacks of root aphid are likely to occur, and insecticide treatment of the soil has often been routinely employed on crops of susceptible varieties as a precaution. However, such treatments are expensive and are usually not very effective (Toscano

et al., 1977), and are often unnecessary because severe root aphid infestations do not occur every year; the production of lettuce varieties with good resistance to this pest has therefore been an important objective.

Dunn (1960) showed that certain lettuce varieties are resistant to attack by *P. bursarius*, but these particular varieties have many characteristics which make them unpopular with growers and consumers. However, recent work by Dunn and Kempton (1974) has shown that some new varieties, particularly Avoncrisp and Avondefiance, are both aphid-resistant and commercially acceptable. These were bred at the National Vegetable Research Station in England, initially for resistance to downy mildew caused by a fungal pathogen, *Bremia lactucae*. Both varieties were derived from a lettuce line, Imperial 45634-M, which originated from California and which is very resistant to root aphid (Dunn, 1960). This resistance, which may have come from a Russian line of *Lactuca serriola* (Dunn, 1977) has been retained during breeding programmes, without deliberate exposure of the breeding material to the root aphid during the generations of selection and multiplication (Dunn, 1974). Resistance to root aphid has also been found in *L. virosa* and *L. saligna* (Dunn, 1977).

To compare the levels of root-aphid resistance of Avoncrisp, Avondefiance and other varieties, three plots of each variety were planted in a randomized block design in a field where natural infestations of *P. bursarius* were likely to occur (Dunn and Kempton, 1974). The number of aphids on the roots of each

Table 11.3 RESISTANCE TO ROOT APHID IN FIVE LETTUCE VARIETIES IN A FIELD EXPERIMENT (AFTER DUNN AND KEMPTON, 1974)

Variety	No. of aphids per plant	Percentage of plants		Undamaged
		Killed	Stunted	
Mildura	6846	87	13	0
Borough Wonder	6014	62	38	0
Webb's Wonderful	5980	31	69	0
Avoncrisp	0	0	0	100
Avondefiance	1	0	0	100

plant was estimated and the extent of damage to the head was assessed. The results (Table 11.3) show that both Avoncrisp and Avondefiance were completely undamaged by root aphids when plants of several other varieties were severely stunted or killed (Figure 11.7).

This extreme resistance derived from Imperial 45634-M is not based on non-preference mechanisms, because approximately equal numbers of alate *P. bursarius* were found on resistant and susceptible varieties in naturally infested field experiments (Dunn and Kempton, 1974). The fact that roots of Avoncrisp and Avondefiance are not colonized by *P. bursarius* (Figure 11.8) seems to be attributable entirely to antibiosis, although there is no published information about the nature of the mechanisms involved. No resistance-breaking biotypes of *P. bursarius* have been reported but resistant varieties have not yet been grown extensively.



*Figure 11.7 Resistant lettuce varieties can give a better control of root aphids (*Pemphigus bursarius*) than any insecticides. In this picture, plants of a susceptible variety (foreground) have been badly damaged or killed, whereas plants of a resistant variety, Avoncrisp, in the background are unaffected. (By courtesy of Dr J.A. Dunn, National Vegetable Research Station, Wellesbourne)*



*Figure 11.8 Under conditions where susceptible lettuce plants (right) become heavily infested with root aphids (*Pemphigus bursarius*), very few aphids are found on the roots of the resistant Avoncrisp (left). (By courtesy of Dr J.A. Dunn, National Vegetable Research Station, Wellesbourne)*

In studying the inheritance of resistance to root aphid, Dunn (1974) crossed Avoncrisp and Avondefiance with two susceptible varieties in all possible combinations, including reciprocals. Experiments on the progenies using apterae of *P. bursarius* showed that resistance is controlled both by cytoplasmic and genetic factors. Although Avoncrisp and Avondefiance are resistant both to the root aphid and to downy mildew, there is no apparent linkage between the genes which control these two types of resistance.

The resistance to root aphids in lettuce varieties derived from Imperial 45634-M is unusual in two respects. First, it is one of the rare examples of resistance to a pest or disease that is inherited cytoplasmically. Second, the resistance is so effective in the field that resistant varieties give a better control of root aphids than do applications of even the most effective pesticides.

Scots Pine

Scots (or Scotch) pine (*Pinus sylvestris*) is a most important timber tree in the northern parts of Europe and Asia where it is native, and it has recently become one of the major cultivated conifer species in North America. Several insect pests can cause serious damage to Scots pine, including the pine root collar weevil, European pine sawfly, Zimmerman pine moth and white (Eastern) pine shoot borer. Varietal differences in resistance to these pests and to a bird pest, the pine grosbeak (*Pinicola enucleator*), have been demonstrated in Michigan (Wright *et al.*, 1976). Two main criteria – the percentage of trees attacked and the average amount of damage per tree – were used to compare the resistance of different varieties.

PINE ROOT COLLAR WEEVIL

This weevil (*Hylobius radialis*) is probably the most destructive pest of young Scots pines in North America. Adult weevils oviposit near the root collar of trees that are more than 1 metre tall and with stems more than 2.5 cm in diameter. Larvae hatch from the eggs to burrow in the cambium near the base of the tree and feed in the inner tissues of the bark. A few larvae can completely girdle a young tree, causing it to die two or three years later. Chemical control of this pest is difficult because the damage remains hidden until infested trees are badly affected.

In trials involving 108 varieties of Scots pine from different areas of its natural habitat, considerable varietal differences in mortality of infested trees were observed (Wright and Wilson, 1972). For example, in six varieties from Scandinavia and Siberia, an average of 31.8 per cent of trees were killed; corresponding figures for five varieties from central Europe and eight from western and southern Eurasia were 57.8 and 20.9 per cent respectively. Percentage mortalities of some individual varieties are given in *Table 11.4*.

The nature of resistance to the pine root collar weevil is not understood. Varieties from southern Europe, which were more resistant than the other varieties tested, had unusually low concentrations of monoterpenes in the oleoresins produced by the inner bark, and of sodium and nitrogen in the leaves. However, no causal relationship has been demonstrated between any

Table 11.4 DIFFERENCES IN SUSCEPTIBILITY TO INSECT AND BIRD PESTS OF SOME SCOTS PINE VARIETIES (AFTER STEINER, 1974; WRIGHT *ET AL.*, 1976)

Type of tree	Percentage of trees attacked by				
	Root collar weevil	European pine sawfly	Zimmerman pine moth	White (Eastern) pine shoot borer	Pine grosbeak
Scandinavian and Siberian varieties					
<i>lapponica</i>	14	0	15	5	77
<i>rigensis</i>	45	6	47	31	30
<i>uralensis</i>	40	3	61	19	25
Central European varieties					
<i>polonica</i>	67	19	62	37	5
<i>hercynica</i>	69	21	57	41	10
<i>carpatica</i>	43	20	62	41	7
West and South Eurasian varieties					
'E. Anglia'	55	27	75	36	28
<i>scotica</i>	31	7	57	41	46
<i>iberica</i>	17	11	33	58	7
<i>armena</i>	12	7	29	51	20

morphological or biochemical characteristics of the host and resistance to *H. radialis*.

Severe infestations of pine root collar weevil are common only on dry, sandy soils. In areas where damage from this pest can be expected, it is important to grow only trees of the more resistant varieties. The considerable natural variation in resistance to *H. radialis* suggests that plant breeders should be able to produce varieties that are even more resistant than those which are currently available.

EUROPEAN PINE SAWFLY

The sawfly *Neodiprion sertifer* can be a serious pest of Scots pine in the USA and heavy infestations can cause complete defoliation of trees in the most susceptible varieties; this defoliation significantly reduces the growth rate of affected trees in the following year. In variety trials in Michigan, Wright *et al.* (1976) found that a much smaller proportion of Scandinavian and Siberian varieties (2.2 per cent) were infested than were those from central Europe (21.2 per cent) or western or southern Eurasia (12.8 per cent). The variety *uralensis* was the most resistant of these varieties in relation to its growth rate and supported fewest sawfly colonies per branch (Table 11.4). This resistance is apparently based on antibiosis, because larvae of *N. sertifer* grew only half as fast on this variety as on the others tested.

The variety *uralensis* has several undesirable commercial attributes, but it should be possible to develop commercially acceptable resistant varieties using *uralensis* as a parent.

ZIMMERMAN PINE MOTH

The destructive pest, *Dioryctria zimmermani*, which is a native of the USA, can seriously damage Scots pine plantations in Michigan. Female moths lay their eggs on the bark in late Summer. Larvae hatch from the eggs within a few days but do not feed until the following Spring, when they tunnel into the cambial region. This feeding can kill twigs and branches, or even whole trees, if several larvae girdle the stem.

Varietal differences in resistance to *D. zimmermani* have been observed in plantations in Michigan (Wright, Wilson and Bright, 1975). Varieties from southern Europe were generally more resistant than those from northern and central Europe (Table 11.4). Resistant trees often contain exceptionally low levels of monoterpenes, particularly 3-carene and terpinolene, in the cortical oleoresin but a direct causal relationship between the concentrations of these compounds and resistance has not been established.

WHITE (EASTERN) PINE SHOOT BORER

The shoot borer *Eucosma gloriola* is native to Canada and the USA and can attack many economically important conifer species. Eggs are laid on the sheaths of new pine needles, and the larvae which hatch from them bore into expanding new shoots in the Spring and feed on the pith. These attacks can kill young twigs and can markedly decrease the growth rate of affected trees. Steiner (1974) reported highly significant differences in resistance to *E. gloriola* among 112 varieties of Scots pine derived from many parts of Europe and Asia. Varieties from southern and western Eurasia were much more susceptible than those from Scandinavia or Siberia (Table 11.4). Resistance was correlated with the latitude or origin of the varieties, with yellow foliage colour in Winter, and with earliness of bud burst, but not with rate of growth.

PINE GROSBEAK

The pine grosbeak (*Pinicola enucleator*) is a serious bird pest of Scots pine in the USA, feeding on the buds during the Winter. Heavy infestations can stunt the growth of trees and deform the stems. Wright *et al.* (1976) recorded the extent of pine grosbeak damage on more than 100 Scots pine varieties in Michigan over a period of some years, and several varietal differences were noted. Most damage was recorded on varieties from northern Europe and Asia and least on fast-growing varieties from central Europe (Table 11.4).

CONCLUSIONS

Although no varieties of *Pinus sylvestris* are completely resistant to any of the insect or bird pests mentioned in this Section, striking differences in resistance to each have been observed in existing varieties. Unfortunately, resistance to each pest seems to be inherited independently and no single variety is resistant to all. Indeed, varieties that are very resistant to one pest are often very susceptible to another (Table 11.4). This means that growers should choose those varieties which are resistant to the pests that are most likely to be troublesome in their area. It should be possible for plant breeders to develop varieties

that are resistant to all the major pests of North America, northern Europe and Asia, although the breeding of new, resistant varieties of Scots pine can be expected to take many years to achieve.

Citrus

CALIFORNIA RED SCALE

The California red scale insect (*Aonidiella aurantii*) is one of the most serious pests of citrus crops in many of the major citrus-growing areas of the world, including the USA, South Africa, Australia, New Zealand, Mexico, Brazil, North Africa, India and China (Cameron, Carman and Soost, 1969). All common citrus varieties are attacked but lemons are usually considered to be particularly susceptible, while mandarins are the most resistant. This pest feeds on the fruit, leaves and branches of citrus, and this can significantly reduce the yield and quality of the fruit. Trees can be killed by heavy infestations of red scale. Insecticides are commonly employed to control California red scale, but these are expensive and do not always give an adequate control. In addition there are potential residue problems with some of the most effective insecticides. For these reasons, the possibilities of breeding for resistance to this pest have been explored, particularly at the California Citrus Research Center at Riverside, California.

Cameron *et al.* (1969; 1975) confirmed experimentally that mandarin is very resistant to red scale and that lemon is very susceptible. Orange and grapefruit apparently show an intermediate level of resistance. The resistance of hybrids between mandarin and orange or grapefruit is intermediate between that of the parents. The parents and hybrids were maintained in a replicated field trial between 1960 and 1974; damage due to California red scale was assessed visually on individual trees in successive years, using 0–5 grading systems based on the extent of branch dieback caused by red scale, or on the number of scale insects present. On each occasion, the ranking for resistance to California red scale was substantially the same; mandarin and hybrids with the most mandarin ancestry had the lightest infestations of *A. aurantii* and suffered the least damage.

The inheritance of resistance is quantitative (Cameron *et al.*, 1969) and resistance is, therefore, presumably controlled by several genes. The factors that are responsible for differences in resistance have not been studied in detail, and more investigations into the nature and inheritance of this resistance are needed. These inherited differences in resistance to California red scale in citrus varieties could probably be used to produce resistant varieties of all the major citrus species.

Sorghum

BIRD PESTS

Sorghum is badly attacked by many species of birds, particularly those which eat the grain, almost wherever the crop is grown. Damage by members of the crow (*Corvus*) and sparrow (*Passer*) families is particularly severe in the USA, Central America and parts of Africa and India. Weaver birds (*Quelea* spp.) are particularly damaging in southern Africa. Bird pests are notoriously difficult

to control, but resistant sorghum varieties will probably significantly reduce bird damage in the future.

Doggett (1957) reported that some African sorghums are damaged much less than others by weaver birds, varieties carrying the gene controlling the 'goose neck' character being particularly resistant. Varieties with 'goose neck' have heads which droop and are therefore less readily accessible to birds. Chetram (1970) reported that two sorghums, known as R1093 and Excel, were less damaged by birds because these varieties had grain which was unpalatable to birds. In the USA, Tipton, Mabbayad and Singletary (1974) reported that Funk BR79, which had been specially selected for bird resistance, usually gave higher yields of grain than other varieties because of its resistance. There was also less grain deterioration and mould formation resulting from bird damage in Funk BR79 than in the other varieties tested; the bird resistance of this variety is associated with a high content of tannins in the grain. Green (1974) also found that the grains of bird-resistant sorghums have a high tannin content, and pericarps that are usually tan or brown whereas pericarps of susceptible varieties are yellow, red, bronze or gold. The *in vitro* organic matter digestibility of grain from bird-susceptible hybrids ranged from 74.5 to 79.8 per cent and from 50.5 to 65.7 per cent in resistant hybrids.

In view of the lower digestibility of the grain of bird-resistant sorghums, Green (1974) suggested that resistant hybrids should be grown only in areas where bird damage is a major problem. This recommendation is supported by the work of Thrasher *et al* (1975), who found that the grains of bird-resistant milo sorghums were small and dark brown, and were less effectively digested by pigs than grains of susceptible varieties.

It is clear, therefore, that bird resistance is generally associated with poor quality and digestibility of the grain in the resistant sorghums that have so far been identified. Nevertheless, there are considerable advantages of bird resistance in terms of grain yield. For instance, Perumal and Sivakumar (1974) showed that losses of grain yield due to bird pests in India exceeded 30 per cent of the potential yield in the most susceptible hybrid, compared with less than 11 per cent in the most resistant variety that was tested.

Although very little is known about the nature or inheritance of resistance, there is considerable scope for breeding bird-resistant sorghums. Particular efforts should be made to overcome the disadvantageous association between resistance and poor quality of the grain.

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RESISTANCE TO PARASITIC WEEDS

The Economic Importance of Parasitic Weeds

Many species of crop plants are subject to attack by parasitic or semi-parasitic flowering plants. About 2500 parasitic seed plant species, belonging to at least 10 families, have been recognized (King, 1966) but fortunately very few of these cause significant agricultural damage. The most important of these belong to a small number of genera in four families. These are *Cuscuta* spp. (dodders) in the Cuscutaceae, *Orobanche* spp. (broomrape) in the Orobanchaceae, *Striga* spp. (witchweeds) in the Scrophulariaceae and *Dendrophthoe* (*Loranthus*) spp. and *Arceuthobium* spp. (dwarf mistletoes) in the Loranthaceae.

The dodders (*Cuscuta* spp.) are common parasites of crop plants and weeds in many temperate regions, and many species can attack several different crop plant species (Ashton and Santana, 1976). Dodder plants consist of pale yellow or orange thin tendril-like stems which branch repeatedly, climbing and sprawling over the aerial parts of their host plants. Where there is a high concentration of dodder seeds in the soil or mixed with the crop plant seed, and when environmental conditions favour the growth of the parasite, a crop can quickly become covered with a dense mat of leafless dodder stems. This can shade the crop plants so that their rate of photosynthesis falls, causing decreases in yields. In addition, dodder extracts water, salts and organic compounds from the host through specialized feeding structures (haustoria) which penetrate the tissues of the leaves and stems of the host plant. Host plants can become seriously debilitated.

Although dodders are very widely distributed in many parts of Europe, Asia Minor and the USA, it is difficult to estimate accurately the amount of damage which they cause. It is clear, however, that this damage is often very significant; for example, dodder is the most serious pest of sugar beet in Turkey and yields of sugar can be greatly depressed by severe attacks. In many other crops damage, or potential damage, is sufficiently great to merit specific control measures (King, 1966).

In contrast to the dodders, parasites of the genus *Orobanche* attack the roots, but not the leaves, of their host plants. They are commonly called 'broomrapes' and are important pests of broad-leaved crop plants, particularly in the warmer and drier temperate and subtropical areas of the world. Nine species

of *Orobanche* are of significant agricultural importance and several of these have wide host ranges (Table 12.1). The most serious crop damage by *Orobanche* occurs in legumes in southern Europe and in tobacco and other Solanaceous crops in India (Kasasian, 1971). Tobacco and tomato crops have also been

Table 12.1 THE HOST RANGE OF SOME AGRICULTURALLY IMPORTANT SPECIES OF *OROBANCHE* (BROOMRAPES) (AFTER KASASIAN, 1971)

<i>Orobanche</i> species	Main crop plant hosts
<i>Orobanche aegyptiaca</i>	<i>Vicia</i> beans, cotton, brassicas, cucurbits, potato, tobacco, tomato
<i>O. cernua</i>	Sunflower, tobacco, tomato
<i>O. muteli</i>	Egg-plant, tobacco, tomato
<i>O. ramosa</i>	Brassicas, cotton, lettuce, hemp, potato, sunflower, tobacco, tomato
<i>O. brassicae</i>	Cabbage, tomato
<i>O. cumana</i> , <i>O. minor</i> , <i>O. crenata</i> , <i>O. lutea</i>	Legumes, sunflower

seriously affected by broomrape in the USA, as have sunflower and hemp in the USSR and Eastern Europe, and tobacco in Turkey. The growth rate and yield of crop plants can be greatly decreased by *Orobanche*, which extracts nutrients, water and salts from the vascular system of the host plant's roots by means of haustoria. Broomrapes are annual plants, which usually flower over a period of several weeks. Each fruiting capsule contains tens of thousands of minute seeds which can be disseminated by wind or irrigation water or by grazing animals. The seeds can remain dormant in the soil for many months.

The genus *Striga* contains many important pests of crop plants in the tropical and subtropical regions of Africa and Asia. *S. asiatica* has also been reported as a pest of maize in the USA, and this species is particularly damaging in South Africa where it is thought to cause greater economic damage than all the known fungal diseases of crop plants combined (Kasasian, 1971). Crops which can be attacked by *Striga* spp. include sorghum, maize, millet, sugar cane, pasture grasses, legumes, tobacco, cucurbits, sunflower and tomato. In certain parts of Tanzania, *S. hermonthea* is an important cause of food shortage in some years and it has been directly responsible for the depopulation of substantial areas of potentially useful agricultural land (Doggett, 1965).

Striga is a root parasite which is completely dependent on its host plant until it emerges above soil level, about six to eight weeks after germination of the seed. The greatest damage to the host plant occurs during this period. Thereafter, the aerial portions of *Striga* produce most of their own nutrients by photosynthesis, but the pest remains dependent on the host for a supply of water and minerals. *Striga* may also cause damage by secreting toxins from the roots, which inhibit the normal growth and development of the host (Hattingh, 1954; Parker, 1976).

The family Loranthaceae includes many important stem parasites of tree crops, including the mistletoes, dwarf mistletoes and species of *Dendrophthoe* and *Loranthus*. Mistletoes (*Phoradendron* spp. and *Viscum* spp.) are semi-parasites whose foliage and stems contain chlorophyll. Different species of

mistletoes can attack apple and pear trees and certain other deciduous trees, including poplar. Although it is unusual for mistletoes to kill infested trees, individual branches can be so badly damaged that they are defoliated, and growth and yield of tree crops can be reduced accordingly. The dwarf mistletoes (*Arceuthobium* spp.), which are near-microscopic parasites of conifers in North America, cause severe damage to pine, spruce and larch plantations. Many infested trees become so deformed that they are worthless as timber and some are eventually killed (King, 1966). The genera *Loranthus* and *Dendrophthoe* contain many important parasites of tropical and subtropical tree crops. For example, *Dendrophthoe falcata* (*Loranthus longiflorus*) is a serious pest of mango and teak in India and can infest a number of fruit-bearing trees including citrus, fig, guava and pomegranate. Related species cause severe damage to rubber trees in Malaya and to kapok in Java (King, 1966).

Some Characteristics of Parasitic Weeds

Dodders (*Cuscuta* spp.) reproduce and spread mainly by seeds, which are of near-microscopic size, although these parasites can reproduce asexually by means of detached pieces of stem. Seedlings live for only a few days if they do not parasitize a suitable host from which they can extract nutrients and water. However, the chances of survival of seedlings in many species of *Cuscuta* are greatly increased by their wide host range. Flowering commences within a few weeks of germination of the seed and is continuous for several months afterwards. Under suitable environmental conditions dodder plants can grow very rapidly, and their tendril-like stems spread from plant to plant.

Spread of *Orobanch*e is also mainly achieved by dispersal of minute seeds, which often contaminate seed of crop plants. These *Orobanch*e seeds can remain viable for more than two years but will usually germinate only in the presence of stimulant substances produced by the roots of host plants and certain other non-host plants. Such stimulants are exuded by roots of pepper, maize, lucerne and clover but, although germination of *Orobanch*e seed is stimulated, the parasite cannot develop functional haustoria in these hosts; it is, therefore, not able to grow or reproduce on these plants, which can accordingly be used as trap crops to decrease the number of dormant *Orobanch*e seeds in the soil. Seeds of *Orobanch*e pass unharmed through the gut of livestock, and cattle and goats, which graze freely on *Orobanch*e shoots and flowers, are responsible for dispersing the seed of this parasite.

Seeds of *Striga* (witchweeds) will, like those of *Orobanch*e, germinate only in the presence of a stimulant substance produced by the roots of host plants and those of a few non-hosts. The microscopic seed of *Striga* has a long dormant period lasting several months, during which germination stimulants are ineffective. Cowpea and soybeans are examples of non-host plants which produce stimulants, and they are used as trap crops in the control of witchweeds.

King (1966) has described the life history of *S. asiatica* (*S. lutea*) in detail and the following is a brief summary of his description. Germination of the seed occurs in the soil wherever root diffusates from a stimulant-producing plant reach a sufficiently high concentration. *Striga* has no root hairs and must absorb all its water and nutrients from the roots of the host through its haustoria.

The aerial parts of *Striga* become green after the parasite emerges from the soil, and it is semi-parasitic thereafter. Flowering starts about one month after emergence from the soil and seeds are formed in capsules, which burst open to disperse the seeds.

Parasites of the Loranthaceae are generally semi-parasites, many of which produce seeds in berries that are attractive to birds, which are largely responsible for their dispersal. In the dwarf mistletoes, however, seeds are enclosed in fruits which dehisce so that the seed is propelled for considerable distances from the host plant. Each seed of these parasites germinates to produce a radicle, which grows into the cortex of the host tree where it swells to form a haustorium. This haustorium grows through the cortex to the cambium, and small root-like structures penetrate into the xylem tissues so that water and nutrients can be withdrawn from the host. During the second year a sprout, which gives rise to green leaves, is formed from the body of the parasite within the cortex.

Agronomic and Chemical Control Methods

Seeds of parasitic weeds, which are usually very small and produced in enormous numbers, are common contaminants of seed of crop plants. These parasites are therefore often unwittingly planted alongside their prospective host plants. It is important to obtain seed only from seed crops which are not infested with parasitic weeds. Where this is not possible, seeds of the parasites should be removed from the crop plant seeds by appropriate seed-cleaning techniques. Dodder seed can be removed from seed of most other plants by using a 'dodder-mill', which consists of rollers covered with felt cloth to which the sticky dodder seeds readily adhere (King, 1966). Such cleaning of seed samples provides one of the easiest and most effective methods of controlling dodder.

Chemicals have been widely used to control most of the economically important parasitic plants, but with varying levels of success. Selective herbicides, including chloroisopropyl phenyl carbamate, have been used extensively to control dodder in alfalfa and 2,4-D or refined diesel oil have been used against *Dendrophthoe falcata*. *Orobanche* has been successfully controlled by sprays of dibromochloropropane, dignat, dinoseb acetate or sodium chloride (Kasasian, 1971). Post-emergence sprays of 2,4-D or MCPA, or pre-planting applications of 2,3,6-TBA or fenac to the soil, have given a good control of *Striga* in a number of different crops. An alternative approach is to fumigate soil that is known to be infested with *Striga* before it is planted with a susceptible crop; methyl bromide has been used on a limited scale for this purpose. The application of artificially produced seed-germination stimulants promises to be an effective method for controlling both *Orobanche* and *Striga*.

Agronomic methods have also been widely practised to control parasitic weeds. For example, trap or catch crops, which produce stimulants that induce seeds of parasitic plants to germinate, have been extensively employed against *Orobanche* and *Striga*. Fungal and insect natural enemies of the weeds can also be used to control them, and Kasasian (1971) cites several examples of successful biological control of *Orobanche* by the fungi *Fusarium* spp. and *Sclerotinia* spp.

Control by Resistant Varieties

Resistant varieties of many crop plant species have played an important part in the control of *Orobanche* and *Striga* but not, apparently, of *Cuscuta* or *Loranthaceae*.

Doughty (1972) reported that early-maturing sorghums are generally less affected than late-maturing varieties, by *Striga hermontheica*. Although there were useful varietal differences in resistance, even supposedly resistant varieties could be badly attacked when *Striga* populations were high. He also showed that maize is usually much more resistant to *S. hermontheica* than sorghum. In a series of field experiments in East Africa, Doggett (1965) found that a sorghum variety, Dobbs, became infested with significantly fewer *S. hermontheica* plants than another variety, Bukura Mahemba (7900 and 91900 per hectare respectively). In other experiments on heavily infested soils, Dobbs carried only 36 per cent as many *S. hermontheica* plants as Bukura Mahemba, and yielded 133 per cent more grain. Dobbs also reduced the number of *S. hermontheica* seeds in the soil and thus benefitted succeeding crops. Doggett concluded that the best control of *Striga* in East Africa will be achieved by growing resistant sorghum varieties, such as Dobbs, together with frequent hand pulling of the weeds.

In India, Rao, Rao and Pardhasaradhy (1967) attempted to develop varieties of sorghum which are resistant to *S. asiatica* (*S. lutea*), because other methods of controlling this pest were too costly. A resistant variety, designated N 13, was released after six years' selection for resistance. Several other Indian varieties of sorghum, including Bonganbilo, YK, Bilichigan, Agyalkodal, Illendi, Nandyal, Mallemari, No 109 and Co 20, are claimed to have a high level of resistance to *Striga* (Kasasian, 1971). Desai, Khatri and Patel (1972), also in India, compared the resistance of eight partially resistant sorghum varieties in different seasons and different environments. They concluded that BC-8 showed the most consistent expression of resistance in different situations and could be expected to give a good control of *Striga* even in heavily infested soils. Resistant varieties of sorghum have been developed in many other countries, including Nigeria, where some lines of dwarf sorghums have shown considerable resistance to *Striga* (Andrews, 1970).

Varieties of pearl millet (*Pennisetum glaucum*) also differ markedly from one another in resistance to *Striga asiatica* (*S. lutea*). For example, Mathur and Bhargava (1971) found that several Indian varieties and breeding lines, including RSJ, showed good tolerance to *Striga*, whereas others, notably RSK and Ghana, were highly susceptible. A male-sterile line, TF 23A, was so resistant in these tests that no *Striga* plants emerged from infested soil in its presence. Many other lines showed intermediate levels of resistance.

Resistance to *Orobanche* spp. has been known to exist for many years in several important crop species including sunflower, brassicas, cucurbits, tomato, tobacco, field beans (*Vicia* spp.) and hemp. There are few published reports about the genetics of resistance to *Orobanche* but the inheritance of resistance to *O. cumana* in sunflower is complex and cytoplasmic inheritance is probably involved (Popov and Lazarov, 1976). Varieties of sunflower resistant to *O. cernua* were developed and distributed in the USSR before 1927 (Pustovoit, 1967, cited in Parker, 1975). Strains of *O. cernua* were later identified which

could attack these varieties, but new varieties with resistance to both the main races of broomrape were soon developed. In heavily infested soils resistant sunflower varieties have often yielded more than seven times as much seed per hectare as susceptible varieties. Resistant varieties are now used on more than 60000000 hectares in the USSR and Eastern Europe and, although attacks of broomrape occur occasionally in some areas, no special control measures are normally necessary (Parker, 1975).

Varietal differences to *Orobanche aegyptiaca* have also been demonstrated in tobacco by Kachan (1966) working in the USSR. Late-maturing tobaccos are generally less affected by broomrape because they flower when the growing season of the parasite has been completed. This corresponds with the situation in clover, where the greatest damage by *O. minor* occurs when the host is flowering (Greenwood, 1952). Tobacco varieties which have shown good resistance to *O. aegyptiaca* in the USSR include Bolgarskii 3056, Kabo 975, Ostrokonets 45 and Trapezond 161. Dalelā and Mathur (1971a) reported that a tobacco variety Vattakppal (Type G) is moderately resistant to *O. cernua* in India, as are a tomato variety (Early Crop) and several eggplant varieties. Răcovia (1973) found significant differences in resistance to *O. ramosa* between tobacco varieties and between species of *Nicotiana* in field and glasshouse tests. *Nicotiana sylvestris*, *N. solanifolia* and *N. paniculata* were particularly resistant to this pest, and Joinar and Hărăgon 226 were among the most resistant varieties.

Orobanche aegyptiaca can reduce the yield of cucumbers by one-half and the sugar content of musk-melons from 8.1 to 4.3 per cent. Several resistant varieties of musk-melons and water melons have been identified by Kabulov and Mukumov (1967). Mukumov (1970) later listed other partially resistant melon varieties and reported that a melon, Kuchka Andizhanskaya and a cucumber, Margelanskii, are completely resistant to *O. aegyptiaca*. Some tomato lines are very resistant to *O. aegyptiaca*, the resistance being dominant and controlled by two or three major and several minor genes (Avdeev and Shcherbinin, 1975).

Resistance to *O. ramosa* has been demonstrated in hemp (*Cannabis sativa*) in the USSR by Senchenko and Kolyadko (1973). When some of the resistant varieties were crossed with a very susceptible variety (Glukhovskaya 10), resistance was dominant in the hybrids. However, some of the resistance was lost after backcrossing to the susceptible parent, suggesting that several genes are involved in the control of resistance.

Kasasian (1973) tested 53 varieties of broad bean (*Vicia faba*) for resistance to *Orobanche crenata* and found that Express was the most resistant. Cubero (1973) found major-gene resistance to *O. crenata* in each of the four main groups of *V. faba* (*major*, *equina*, *minor* and *paucijuga*), but modifying genes seem also to be implicated. The expression of resistance can be greatly affected by environmental conditions, and is decreased particularly in dry soil and dry air.

About 50 varieties of rape (*Brassica campestris*) and mustard (*Brassica juncea*) were tested for resistance to *O. aegyptiaca* in heavily infested, light sandy soils in India by Dalelā and Mathur (1971b). BR 13, BR 40 and M-1 were the most resistant varieties of *B. campestris* and R-2-62, R-28, 428-62 and 488-62 were the most resistant mustard varieties. Most of the other varieties tested were moderately resistant to broomrape, but a few were very susceptible.

Methods of Testing for Resistance

Field, glasshouse and laboratory tests have all been used to compare the resistance of varieties and breeding lines to parasitic weeds. Doggett (1965) evaluated the resistance of different sorghum varieties in field experiments conducted under a wide range of naturally occurring levels of *Striga hermontheica* infestation. Field tests in heavily infested soil were also employed in testing the resistance to *Orobanche aegyptiaca* in rape and mustard (Dalelă and Mathur, 1971b) and to *O. ramosa* in hemp (Senchenko and Kolyadko, 1973). In selecting for increased resistance to *O. ramosa* in hemp, a moderately susceptible variety, Yu S-9, was grown in infested soil; plants that were not badly attacked by broomrape were selected for seed production. The proportion of lightly infested plants in this variety was increased from less than 1 per cent to more than 16 per cent by two cycles of selection. This suggests that it should be possible to develop highly resistant varieties of hemp by recurrent selection.

Techniques of artificially inoculating field plots have also been employed in testing as, for example, with resistance to broomrape in tobacco (Kachan, 1966). About 500 seeds of *O. aegyptiaca* were placed under each tobacco plant when it was being transplanted into the field. Plants that were not badly damaged by broomrape were allowed to interpollinate, and progenies were subjected to further selection tests. This kind of selection resulted in tobacco populations with a greatly increased level of resistance after a few generations of selection.

Many tests of resistance to parasitic weeds have been carried out in pots in the glasshouse or laboratory. For example, Mathur and Bhargava (1971) tested the resistance of several pearl millet varieties to *Striga* in the glasshouse using soil to which *Striga* seeds had been added. The number of *Striga* plants that emerged from the soil in each pot was used to compare the resistance of different varieties, and the vigour of infested plants was also compared. Parker, Hitchcock and Ramaiah (1977) grew batches of sorghum seedlings in sand culture for one week, after which the effects of their root exudates on species of *Striga* were compared. Plants which produced large amounts of compounds that stimulate germination of *Striga* seed were discarded. Răcoviia (1973) studied the effects of root exudates from different *Nicotiana* spp. and tobacco varieties on *Orobanche ramosa* in laboratory tests in search of varietal differences.

There are many advantages in testing for resistance to parasitic weeds in the laboratory or glasshouse, where experimental conditions can be controlled or standardized to some extent. Nevertheless, Doughty (1972) found that laboratory tests of the germination of *Striga* seed in the presence of different host plants sometimes gave a misleading picture of the resistance of these plants in the field. This is probably because resistance is often based on several different factors, all of which may not be expressed in small plants grown under artificial conditions. It is important, therefore, to check the results of glasshouse and laboratory experiments with those of field tests under conditions of natural infestation whenever possible.

The Nature of Resistance

Resistance to *Striga* seems to take the three following forms (Parker *et al.*, 1977): (1) lack of production, by the host, of a substance which stimulates germination

of *Striga* seed; (2) some form of mechanical resistance which prevents penetration by the haustoria; (3) some form of incompatibility between host and parasite, which results in the weak growth of the parasite after it is attached to the host plant.

Williams (1959) reported that a sorghum variety, Framida, was resistant to *Striga hermonthea* because its roots produced very little seed-germination stimulant. Doggett (1965) showed in a series of field experiments that Framida stimulated less germination of *Striga* seed than Dobbs (a resistant variety) or Bukura Mahemba (a very susceptible variety). This showed that the resistance of Dobbs was attributable to factors which operated after germination of *Striga* seed had occurred. The resistance of Dobbs seems to be caused by some kind of incompatibility mechanism and may be partly attributable to a greater thickness of cell walls in the root, which may hinder penetration by the parasite.

Work by Panchenko and Antonova (1974) in the USSR, cited by Parker (1975), has provided information about the mechanisms of resistance to *Orobancha cumana* in sunflower. Seed germination and initial penetration by the parasite into the roots is similar in resistant and susceptible sunflowers. When the haustorium reaches the xylem of resistant sunflowers, however, there is a rapid lignification and thickening of the cell walls in the xylem tissues which apparently prevents the penetration of the stele by the parasite. The parasite is therefore not able to withdraw water and nutrients from the host in sufficient quantities to support the growth of *O. cumana*, and the parasite eventually dies. No such lignification occurs in susceptible sunflowers, and a compatible relationship between host and parasite is established. Although lignification may account for the resistance of many sunflower varieties to *Orobancha*, the existence of other types of resistance, for example a failure to produce a seed-germination stimulant, cannot be discounted.

Genetic Variation in Parasitic Weeds

Although some species of parasitic weeds have wide host ranges, there is ample evidence of physiologic specialization in these parasites which results in a specific relationship between individual genotypes of host and parasite. For example, in the Sudan, sorghum millets are very susceptible to one strain of *Striga hermonthea* to which *Pennisetum* millets are immune. Conversely, *Pennisetum* millets are susceptible to another strain of *S. hermonthea* to which sorghum millets are resistant (Wilson-Jones, 1955). Two distinct races of *S. hermonthea* have been identified in West Africa, one primarily on pearl millet and the other on sorghum (King and Zummo, 1977). Populations of *Striga* that mainly attack maize in Cameroun may belong to a third race. However, there seems to be no clear evidence for the existence of resistance-breaking variants of *Striga* that would be capable of attacking previously resistant varieties of any crop.

Resistance-breaking races of *Orobancha cumana* have been encountered in sunflower. In the late 1920s resistant sunflower varieties failed to give a good control of broomrape in parts of the USSR. Subsequent work showed that this 'breakdown' in resistance was due to the presence of a virulent 'broomrape B' strain of *O. cumana* which could attack previously resistant varieties (Pustovoit, 1967). Further breeding and re-selection for resistance resulted in the development of varieties which are resistant to both of the main strains of *O.*

cumana. Another virulent strain of broomrape, designated the Kishinev race, has since appeared in Moldavia and the Ukraine, and this can attack a previously immune sunflower variety, VNII Mk 1646 (Pogorletskii, 1971). Hybrid sunflowers have been produced which are immune to the new Kishinev race and to the more common Odessa race.

This work has shown that parasitic weeds can become adapted so that they are able to attack some previously resistant varieties. Resistance-breaking strains are, therefore, a potential threat to resistant varieties. Nevertheless, this threat has not materialized to such an extent that the advantages of using resistant varieties in controlling parasitic weeds have been nullified, or even seriously reduced, for significant periods.

Conclusions

Although the effort which has been devoted to breeding for resistance to parasitic weeds has been very limited, the breeding work has generally been very successful. Resistant varieties to many species of *Striga* and *Orobanche* have been developed in several important crop species, and form the main basis of control. In the USSR, for example, varieties of sunflower that are resistant to *Orobanche* occupy over 60000000 hectares, and no other measures to control broomrape are usually necessary (Pustovojt, Plytnikova and Gubin, 1976). Resistant varieties of sorghum have helped greatly to reduce damage by *Striga* spp., particularly in Africa and India. Nevertheless, these parasites still cause considerable losses in some crops in many parts of the world, and an increased effort to breed high-yielding, locally adapted varieties with improved levels of resistance is clearly justified. The potential benefits of breeding for resistance to *Striga* and *Orobanche* are still very considerable.

It is, perhaps, strange that so little work has been done on breeding for resistance to other parasitic weeds, notably the dodders and those of the Loranthaceae, particularly as they are widely distributed and damaging parasites of so many important crop plants and forest trees. A search for sources of resistance to these parasitic weeds and the development of resistant varieties is long overdue.

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THE PRESENT POSITION

Experience with Fungal Diseases

Resistance to fungal diseases has been one of the most important objectives for breeders of most crops. In many instances, disease resistance has been even more important than the improvement of yield and quality, as with the resistance of wheat to stem and leaf rust in North America. However, the use of resistant varieties has usually been only one part of an integrated control programme as in the late blight and wart diseases of potatoes, where resistant varieties have been used in conjunction with fungicides and legislation respectively. With some diseases, such as maize rust and sugar beet downy mildew, most current varieties express enough 'field' resistance for additional control measures to be unnecessary; in such cases, the plant breeder must ensure that this level of resistance is maintained in future varieties.

Resistant varieties have successfully controlled many fungal diseases in most of the major agricultural and horticultural crops. Indeed, several diseases cause very little damage today mainly because most of the varieties that are grown show a high level of durable resistance; there have been no problems with resistance-breaking races of the causal pathogens with these varieties. There is a serious danger that some of this durable resistance will be lost if plant breeders start to use types of resistance which involve hypersensitivity conditioned by major genes; although such resistance would confer near-immunity to disease and be easy to manage in a breeding programme, it would probably be race-specific. This danger exists today in maize where the use of major-gene resistance has been advocated to control rust (*Puccinia sorghi*), although this disease can already be adequately controlled by the durable, partial resistance present in many existing varieties (see page 116). Major-gene hypersensitivity usually masks the expression of other types of resistance, thereby precluding selection for these other types. In consequence, many varieties that express hypersensitivity to a pathogen lack the 'background' resistance to disease that is shown by others and have eventually been damaged by resistance-breaking races as a result. Plant breeders should, therefore, be very cautious about using types of resistance which prevent the maintenance or improvement of other types.

Resistance to some fungal diseases has been durable even when it is known to be race-specific. For example, the resistance of the rice variety Tatep to

Piricularia oryzae (blast disease) is race-specific, but this variety has usually not been badly attacked by the pathogen in the field, although it has been exposed to attack for many years in several countries (*see* page 111). The resistance of the wheat variety Selkirk to stem rust has also been durable though race-specific (*see* page 92), as was the resistance of the tomato, Vagabond, to leaf mould (*see* page 134). The race-specific resistance of the wheat variety Kawvale to loose smut has been effective in the USA since 1932 (Caldwell, 1968).

In many crop species, both race-specific and other types of resistance are known to exist, either together in a single variety or in different varieties. For example, the resistance to yellow rust of certain wheat varieties has been durable whereas the resistance of others has proved to be highly race-specific and transient (*see* page 85). Race-specific and non-race-specific types of resistance to late blight have also been found in potato varieties (*see* page 117) and a similar situation exists in sugar beet varieties that are resistant to downy mildew. Durability or transience of resistance is not, therefore, a feature of particular crop species or varieties.

The capacity of all fungal pathogens to vary genetically seems to be sufficient to enable them to produce resistance-breaking races which can overcome certain types of resistance, when the selection pressure in their favour is great enough. Sexual reproduction is not necessary for the production of these races, and pathogens such as *Puccinia striiformis*, which do not have a sexual stage, have produced many resistance-breaking variants, presumably by mutation or somatic recombination of genes. Durability of resistance is therefore not limited to particular pathogens or groups of pathogens with particular breeding systems.

It appears, therefore, that durability of resistance is not a function either of particular hosts or of particular fungal pathogens, but of interactions between pathogens and certain types of resistance that are expressed by some host plants but not by others. Although there are no clear-cut relationships between any individual types of disease resistance and durability, the following conclusions can be drawn from the examples described in Chapter 4:

- (1) Resistance-breaking races of fungal pathogens have been more common with types of resistance controlled by major genes than with other types. However, some combinations of major genes, often in association with minor modifying genes, have conferred durable resistance in the varieties which carry them, as in the resistance of the wheat variety Selkirk to rusts and of the rice variety Tatep to blast.
- (2) Resistance involving hypersensitivity has often proved to be race-specific. It is difficult to differentiate between the effects of control by major genes and hypersensitivity, however, because hypersensitivity is usually controlled by a few genes of large effect. It is, therefore, reasonable to assume that any hypersensitive response of a host plant that is conditioned by major genes will be more race-specific than other types of resistance.
- (3) Problems with resistance-breaking races have frequently been encountered with resistance derived from wild relatives or from varieties of related species; such resistance is usually controlled by major genes and often involves hypersensitivity.
- (4) Although resistance that is controlled by major genes is easy to use in breeding programmes, complex polygenically inherited types of resistance have also been successfully exploited in very many breeding projects.

- (5) It is not necessary to understand either the genetics or the nature of resistance in order to breed successfully for resistance to fungal diseases. Nevertheless, there is an urgent need to identify those resistance mechanisms which confer durability of resistance, whether they are based on the morphology, anatomy, biochemistry or physiology of the host plant; they can then be positively selected for in breeding for resistance.
- (6) Methods of increasing the durability of resistance which is known to be race-specific should be investigated more thoroughly. Such methods include the use of multiline varieties or mixtures of varieties (Frey, Browning and Simons, 1977), diversification of varieties and combinations of resistance genes that are not easily matched by corresponding combinations of virulence genes in the pathogen. It is unlikely, however, that recycling of resistance genes, involving the temporary withdrawal of varieties in which resistance has been overcome by new races of a pathogen, will significantly improve the durability of resistance controlled by those genes. This was the experience in Australia with stem rust of wheat, when Eureka was withdrawn from cultivation for several years before it was reintroduced (*see* page 93).
- (7) Combinations of resistance genes, which control similar resistance mechanisms (for example, hypersensitivity) have not usually conferred durability. However, combinations of different types of resistance, for example disease escape, hypersensitivity and tolerance, have usually been durable.
- (8) Although the genetic variability of fungal pathogens has led to the breakdown of resistance in many varieties, there are very few examples of catastrophic losses as a result of such breakdowns. Breeding for resistance has generally been very successful in reducing damage caused by most of the major fungal diseases. Although much higher levels of durable resistance can probably be achieved, even partial resistance can give a very effective disease control; it may, however, sometimes need to be supported by other control methods. For example, it may be necessary to apply fungicides to partially resistant varieties under conditions which favour the development of disease epidemics, but such applications can be expected to give a much better control on these varieties than on susceptible varieties.
- (9) It is not necessary to breed varieties that are so highly resistant that they appear immune. The breeding of varieties with an intermediate level of durable resistance, which gives an adequate level of disease control in most situations, should therefore be the main objective in breeding for resistance to fungal diseases. This objective would allow plant breeders to concentrate their efforts on selecting for improved yield and quality in the absence of disease, and less time would need to be spent on breeding varieties with complete resistance to new races of pathogens.

Experience with Bacterial Diseases

Varieties that are resistant to important bacterial diseases have been developed in many species of crop plants. Although these varieties have helped to reduce damage from disease, they have not usually been the main method of control. For instance, chemical seed treatments have been very important in the control of bacterial blight of cotton, wildfire disease of tobacco and bacterial canker of tomato, although resistant varieties have been available. Eradication of diseased

trees has been the main method of controlling fireblight of pears, and cultural practices have been important in the control of bacterial blight of rice and the Granville wilt and wildfire diseases of tobacco.

It should be possible for plant breeders to develop varieties with higher levels of resistance, and these could play a much greater part in the control of bacterial diseases. Much less is known about the nature of resistance to bacteria than to fungal pathogens. Although hypersensitivity is often involved, and resistance is frequently controlled by major genes, resistance to many bacterial diseases has been durable even if race-specific. Plant pathogenic bacteria, unlike fungal pathogens, are extracellular parasites; hypersensitivity to biotrophic fungi may, therefore, involve quite different biochemical and physiological mechanisms to those involved in hypersensitivity to bacteria. The genetics of resistance is often complex, involving combinations of major and minor genes; this suggests that the resistance mechanisms that they control may be correspondingly more complex than is the case with monogenic resistance to some fungal pathogens.

The expression of resistance to bacterial diseases is often very labile, being greatly influenced by changes in environmental conditions. This lability can hinder investigations concerning the nature and genetics of resistance, and can complicate selection for resistance, but it may contribute in some way to the durability of resistance. Although resistance to many bacterial diseases is known to be race-specific, the resistance has usually been durable and there have been few serious breakdowns of resistance in the field.

Experience with Virus Diseases

Resistance to some virus diseases has been of supreme importance in several crops. For example, resistance to curly top virus is an essential prerequisite of sugar beet varieties grown to the west of the Rocky Mountains in the USA. Resistant varieties are the main method of controlling several important virus diseases, including tungro in rice, leaf curl in cotton and curly top in tomato. In general, however, resistance is just one of several control methods, including the chemical control of insect vectors, and cultural practices, such as the removal of potential sources of infection, and appropriate crop rotations. There has recently been increased interest in breeding for resistance to virus diseases in many crops, because pesticides are becoming increasingly expensive and insecticide-resistant forms of many vectors have become prevalent.

The examples described in Chapter 9 show that virus-resistant varieties have reduced damage from virus diseases in many crops. However, resistant varieties, such as curly top-resistant varieties of sugar beet, can still be seriously damaged when infected with virulent virus strains, and greater levels of resistance to such diseases are clearly desirable. Many resistant varieties have not been used extensively even when they have been available for several years, presumably because they are less productive or of lower quality than susceptible varieties. Thus, resistance to tobacco mosaic virus in tomatoes is frequently associated with low yield (*see* page 257). A sugar beet variety, *Maris Vanguard*, which is tolerant to virus yellows, has not been grown on a large scale mainly because it is multigerm (*i.e.* it has fruits with several seeds) and yields roots of low processing quality in the absence of virus infection (*see* page 240). These examples emphasize that resistance to diseases is only one of many breeding

objectives, and that resistant varieties are unlikely to be used extensively if they have many undesirable features.

Resistance to several virus diseases has been durable in varieties that have been grown on a large scale for many years, such as potato virus X (*see* page 237), sugar beet curly top (*see* page 246), aphid-transmitted virus diseases of raspberry (*see* page 260) and leaf curl of cotton (*see* page 263). Resistance to many other viruses, including hoja blanca and tungro in rice, tobacco mosaic in tobacco and virus yellows in sugar beet, seems also to be durable although resistant varieties have not yet been grown extensively. Resistance to a few diseases has been highly race-specific, as in tomato mosaic (*see* page 257), but resistance-breaking virus strains have generally been much less troublesome to the plant breeder than have variants of fungal pathogens.

The reasons for the greater durability of resistance to virus diseases are not understood. Hypersensitivity is involved in the race-specific resistance to tomatoes to tobacco mosaic virus and to several viruses in other crops. However, the hypersensitivity to virus X expressed by some potato varieties, including King Edward and Epicure, is as effective today as it was more than 50 years ago (*see* page 237). This shows that hypersensitivity is not always associated with transient resistance to virus diseases.

Several distinct types of resistance to virus diseases have been identified. Resistance to virus infection has been found in many crops, including potato, sugar beet, rice, tobacco, cocoa, raspberries, barley and tomatoes. Extreme resistance (which by definition is effective against all strains of a virus) has also been used a great deal by breeders, particularly in potatoes and tomatoes. Many resistant varieties express resistance to virus multiplication, and resistance to the vectors of viruses is also common. Some of these resistance components are controlled by major genes, others by polygenes. For example, resistance of potatoes to potato viruses X and Y can involve extreme resistance (controlled by major genes), hypersensitivity (controlled by different major genes) and resistance to virus infection (polygenically controlled). Resistance to aphid-transmitted viruses in raspberry can be based on virus avoidance, virus tolerance and resistance to the aphid vectors, and each of these resistance components is under separate genetic control (*see* page 260). Resistance to many other virus diseases, including swollen shoot of cocoa (*see* page 255), virus yellows and curly top of sugar beet, rice tungro disease and potato leaf roll, is complex and involves several independently inherited types of resistance.

This complexity of resistance may be partly responsible for the observed durability of resistance to many virus diseases, and breeders should therefore try to incorporate several different types of resistance into their varieties, as suggested by Russell (1972). Combinations of virus resistance and vector resistance are probably particularly worth while because it is unlikely that resistance-breaking forms of both virus and vector would simultaneously become widespread.

An alternative explanation for the durability of resistance to virus diseases is that viruses may be less genetically variable than fungal pathogens. However, the theoretical capacity for genetic variation in viruses is very great and it is known that there are many variants of most plant viruses. These variants are differentiated according to their various biological and biochemical properties, but not usually by their interactions with different host genotypes. Strains of tobacco mosaic virus have developed which match specific resistance genes in

the tomato, and there is no reason to suppose that this virus is more variable than other viruses. It seems probable that all viruses are capable of producing resistance-breaking strains with some types of resistance, such as hypersensitivity, but not with others.

There are very good prospects of developing varieties with a high level of resistance to most viruses in all the major crop plants. Good sources of resistance are generally present either in cultivated varieties or in related wild species. Several different types of resistance to most viruses are available to the plant breeder and, as has already been suggested, combinations of different types can be expected to be more durable than single types. All types of resistance to viruses, including virus tolerance, and to the vectors which transmit them, can contribute to disease control in the field. Many of the theoretical disadvantages of virus tolerance (*see* page 225) have not been encountered in practice, and tolerant varieties have played a very significant part in reducing damage from virus diseases in many crops.

Although there are good reasons for breeding for complex types of resistance, there are also many advantages in keeping resistance as simple as possible. Extreme resistance, which has been used in potatoes against potato viruses X, Y and A, is effective and is very easy to manage because it can easily be selected for and is controlled by major genes; only two resistance genes are necessary to confer extreme resistance to all three viruses. Although simply inherited types of resistance are often highly race-specific, there are no indications of resistance-breaking virus strains in the case of extreme resistance to potato viruses X, Y and A. It is very unlikely that any strain of one of these viruses would be able to overcome the broad-spectrum resistance mechanism that is involved.

Although many different types of resistance have been characterized and the genetics of resistance is at least partly understood in virus diseases of several crops, breeding for resistance to other diseases has been successfully accomplished without this kind of detailed knowledge. For example, an empirical approach to selection for resistance to curly top in sugar beet and leaf curl in cotton has given excellent results, and the varieties so produced have continued to give a worth-while disease control for many years.

Experience with Animal Pests

There have been many striking successes in breeding for resistance to animal pests, particularly with invertebrate animals. Animal pests tend to be most damaging to crop plants in warm temperate, subtropical and tropical climates and it is to be expected, therefore, that the greatest advances in breeding for resistance to such pests would have been made in crops grown in warm countries. Much of this work has been pioneered in the USA, where many resistant varieties have been developed.

Resistant varieties of crop plants have played an important part in controlling many insect, mite and nematode pests, and there are good prospects of developing varieties resistant to some vertebrate pests, including birds. Several pests, such as wheat stem sawfly and cereal leaf beetle in wheat and jassids in cotton, have been adequately controlled solely by the use of resistant varieties. Resistant lettuce varieties give a better control of root aphids (*Pemphigus bursarius*) than can be achieved with any pesticide.

More often, however, resistant varieties give only a partial control of a pest, and additional control measures must be adopted in severe infestations. Even partial resistance can be very valuable, however, because other control measures are generally much more effective on partially resistant varieties than on susceptible varieties. For example, fewer applications of insecticides are usually needed to control insect pests on resistant varieties than on susceptible varieties. Nevertheless, frequent applications of insecticides are necessary even on resistant varieties, to achieve an adequate control of some pests, including leafhoppers on rice (*see* page 344) and earworms on maize (*see* page 350).

Parasites and predators can also give a more effective control of insect pests on resistant than on susceptible varieties. For instance, the European corn borer is more effectively controlled by a Protozoan parasite, *Nosemia pyrausta*, on partially resistant maize varieties than on susceptible plants (*see* page 350). A combination of crop rotation and resistant varieties gives a very good control of the potato cyst nematodes. Resistant varieties can therefore play an important part in integrated pest control programmes even when they do not express high levels of resistance. However, pesticides are often too expensive to use on low-value crops and pesticide-resistant forms of many pests have become prevalent; it is, therefore, often very desirable to develop resistant varieties that do not need to be supported by chemical control measures.

Table 13.1 SOME EXAMPLES OF DURABLE AND TRANSIENT RESISTANCE TO ANIMAL PESTS

Crop	Pest	Type of resistance		
		Probably durable	Not known	Probably transient
Cotton	Boll weevil	+		
	Bollworm		+	
	Jassid	+		
Rice	Stem borers	+		
	Leafhoppers*			+
Maize	European corn borer	+		
	Earworm	+		
	Leaf aphid		+	
Alfalfa	Spotted aphid	+		
	Pea aphid*	+		
Potatoes	Aphids	+		
	Cyst nematodes*			+
Wheat	Hessian fly	+		
	Greenbugs	+		
	Stem sawfly	+		
	Leaf beetle	+		
Barley	Cyst nematodes*		+	
Oats	Stem nematode	+		
Clover	Stem nematode	+		
Raspberry	Aphids*	+		
Sugar beet	Aphids		+	
Lettuce	Root aphid		+	
Brassicas	Cabbage aphid*			+

*Resistance is probably race-specific

Resistance-breaking variants of animal pests have not usually been a serious problem for the plant breeder and most resistance to pests has been durable (*Table 13.1*). Resistance to several pests, for example the wheat Hessian fly, has been durable in spite of the fact that it is race-specific. With many other pests, no specific interaction has been observed between individual genotypes of pest and host plant; for instance, the resistance of wheat to stem sawfly or cereal leaf beetle is apparently non-race-specific. Race specificity has not been of major importance with many pests.

Durability does not seem to be associated with any particular type of resistance. Most of the resistance-breaking problems have arisen with types of resistance that involve some sort of antibiosis, but there are even more examples of durable resistance that is based on antibiosis. Some kinds of durable resistance are controlled by major genes while others are controlled by minor genes or polygenes; the genetics of resistance therefore apparently bears little relation to the durability of resistance. There is some indication, however, that resistance introduced into a crop species from a wild relative is more likely to be more race-specific and possibly less durable than that from intraspecific sources. Insects have a great capacity for genetic variation, as shown by their ability to produce insecticide-resistant forms. It is unlikely, therefore, that lack of genetic variability would be responsible for the greater durability of resistance to insect pests. It is true that populations of animal pests are generally smaller than those of fungal pathogens and there is therefore less potential for genetic variation within a population, but this has not hindered the development of insect variants adapted to overcome adverse conditions. The reasons for the greater durability of resistance to invertebrate pests are not understood, but this situation is a happy one for the plant breeder.

Breeding for resistance to insect and nematode pests has been outstandingly successful in most crops where resistance to pests has been an important breeding objective. Although resistance to invertebrate pests has usually given only a partial control, and has often had to be supplemented by other control measures, the resistance has generally been durable. Resistance to vertebrate pests is more difficult to achieve and has been attempted on a large scale only recently. However, the successes which have been achieved with bird-resistant varieties of sorghum and maize in the USA, should encourage plant breeders everywhere to explore the possibility of reducing bird and rodent damage by resistant varieties. Breeding for resistance to most pests is a long and tedious process which can involve selecting and testing for small, quantitatively inherited improvements in resistance. However, the release of the alfalfa variety Cody, which is highly resistant to the spotted alfalfa aphid, within five years of the recognition of this aphid as a serious pest, should inspire plant breeders everywhere to search intensively for new sources of resistance to pests.

The Importance of Parasite Variability

Wolfe (1972) has suggested that the force of the evolutionary direction exerted on major pathogens of cereals by the growing of highly resistant varieties has frustrated many attempts to breed for stable resistance. This is because virulent, resistance-breaking forms of the pathogens can multiply freely without competition on the varieties which they alone can attack. This situation has occurred

frequently with several fungal diseases, for example powdery mildew of barley (see page 104). The level of resistance to the brown planthopper is so high in some rice varieties (see page 346) that populations of this insect have been forced to change genetically, so that they can attack these varieties, or be eliminated (Khush, 1977). Resistance of rice varieties to the green leafhopper is much less extreme and there is therefore less selection pressure in favour of resistance-breaking biotypes. These differences in selection pressure are reflected in the durability of resistance to these two insect pests in rice; the resistance to brown planthoppers of some rice varieties has quickly been broken down by new biotypes, whereas most resistant varieties have continued to give a good control of the green leafhopper for many years. These and several other examples strongly indicate that the dangers of parasite variation to the plant breeder are greatest when the expression of resistance in the host plant is very high.

Van Der Plank (1968) predicted that, in the absence of continuing selection pressure in favour of resistance-breaking variants, pathogen populations will tend to lose unnecessary virulence genes, because complex variants carrying several resistance genes are less fit to survive and reproduce than other variants. This concept of stabilizing selection has been challenged by several workers. Although Wolfe (1972) found that there is a low frequency of complex races of *Erysiphe graminis* on wheat and barley in the absence of positive selection in their favour, he attributed this to random chance rather than to stabilizing selection. Complex races of many fungal pathogens are as fit as simple races and often carry unnecessary virulence genes (Crill, Jones and Burgis, 1974). Mackey (1973) identified 10 virulence genes in a population of *Puccinia graminis* which had no race-specific functions in the environment in which that population was collected. This lack of stabilizing selection in *P. graminis* was probably responsible for the second rapid breakdown of resistance in Eureka when this wheat variety was re-introduced into cultivation in Australia after an absence of several years (see page 93).

Although all parasites of crop plants seem to have a very considerable capacity for genetic variability, there is a danger of introducing new resistance-breaking variants of pathogens into areas where they have not occurred before, particularly in isolated areas such as islands, even when other variants are already present. For example, if the New Jersey strain of tomato spotted wilt virus had been introduced into Hawaii in the mid-1950s, susceptible local tomato varieties might have been severely damaged (see page 259).

The Genetics of Host-Plant Resistance

Several different kinds of resistance to most of the important parasites of crop plants are known. Some of these types are controlled by major genes, others by minor genes and still others by combinations of major and minor genes. There has been much discussion about the relative advantages and disadvantages of using resistance controlled by major genes or polygenes in breeding for resistance (e.g. Nelson, 1973).

There are many advantages of using monogenic resistance to pathogens and pests: (1) monogenic resistance is easy to manage in a breeding programme because resistance reactions are quite obvious and can be detected by simple

tests; (2) the resistance usually gives a complete control of disease in the field; (3) the reproduction and spread of a pathogen is generally completely curtailed (Crill, Jones and Burgis, 1974). Crill *et al.* (1974) considered that the development of agronomically acceptable varieties with polygenically controlled resistance to each of several diseases would be impracticable because of the very great facilities and resources which would be necessary. It is to be expected, therefore, that most of the examples of multiple resistance to pests and diseases mentioned in this book involve simply inherited types of resistance, for example to the smut diseases of sorghums (*see* page 138), to the *Fusarium* and bacterial wilts of tomato (*see* page 191) and to fungal and bacterial diseases and insect pests of rice (*see* page 347). Nevertheless, some varieties of crop plants express a polygenically controlled, intermediate level of resistance to several pests and diseases which is sufficient to prevent heavy attacks in most situations. For example, several sugar beet varieties are moderately resistant to downy mildew (*Peronospora farinosa*), powdery mildew (*Erysiphe betae*), *Ramularia* leaf spot (*Ramularia beticola*) and rust (*Uromyces betae*) and therefore they have not been badly attacked by any of these diseases (Russell, 1969). Thus, it is not impossible to achieve a high level of polygenically controlled resistance to several different diseases in a single variety.

The expression of resistance controlled by major genes is often increased by the presence of minor, modifying genes. The level of resistance to apple scab (*Venturia inaequalis*) conferred by the *Vf* gene is increased by the action of cumulative minor genes which can be contributed by either susceptible or resistant parents (Rousselle, Williams and Hough, 1974). The resistance of apples to collar rot and powdery mildew is also conditioned by major genes, the effects of which can be modified by minor genes (Alston, Watkins and Wertz, 1974). Such combinations of major and minor genes may reduce the possibility of resistance-breaking situations. However, it is often difficult to test for the presence of minor genes in the presence of masking major genes.

The genetics of resistance to many pests and diseases is very complex and is therefore not properly understood. There is often considerable disagreement about the number of resistance genes involved, even where major genes are concerned, as in resistance to bacterial blights of cotton (*see* page 180), rice (*see* page 185) and tobacco (*see* page 188). Such confusion is often caused by the fact that the expression of resistance can be very labile, being greatly influenced by changes in environmental conditions. For this reason, more detailed investigations of the genetics and expression of resistance to pests and diseases under precisely controlled conditions are urgently needed.

The expression and durability of host-plant resistance to a parasite depends to a large extent on the types of resistance mechanisms which operate in the host and on the kinds of mechanisms in the parasite which might be able to circumvent the defence system of the host. As Sidhu (1975) points out, it is the **physiological** basis of resistance that is important in host-parasite relationships. Resistance to a parasite will be race-specific, whether it is conditioned by major genes or polygenes, if some variants of that parasite are able to overcome the resistance mechanism involved. If the ability to circumvent a resistance mechanism is outside the range of genetic variability of a parasite, no resistance-breaking variants are possible and the resistance will be truly non-race-specific. Race-specificity is, therefore, not a question of a virulence gene in a parasite matching a corresponding resistance gene in the host, but rather of an 'attack'

system of the parasite proving more than a match for a particular defence system. The complexity of the resistance mechanisms is, therefore, a major factor in determining whether resistance will be durable or transient. A single gene codes for a specific protein, which is often an enzyme, and resistance controlled by a few major genes therefore inevitably involves simple resistance mechanisms. Polygenic control of resistance implies that several proteins are involved in a series of complex metabolic interactions which together constitute one or more resistance mechanisms; such complex mechanisms are presumably more difficult for parasites to overcome than are simple mechanisms, and this may help to explain the greater race-specificity of major-gene resistance.

Table 13.2 SOME EXAMPLES OF DURABLE MAJOR-GENE RESISTANCE TO PESTS AND DISEASES

<i>Crop plant</i>	<i>Pest or disease</i>	<i>Reference</i>
Cucumber	Cercospora leaf spot (<i>Cercospora</i> spp.).	Day (1968)
Sorghum	Milo disease (<i>Periconia circinata</i>)	see page 138
Cotton	Bacterial blight (<i>Xanthomonas malvacearum</i>)	Innes (1974)
Cotton	Leaf curl virus	Siddig (1970)
Cotton	Jassids (<i>Empoasca</i> spp.).	Arnold (1969)
Raspberry	<i>Botrytis cinerea</i>	Knight and Keep (1958); Jennings (1962)
Raspberry	Aphids (<i>Amphorophora</i> spp.).	Keep <i>et al.</i> (1970); Daubeney (1972); Kennedy <i>et al.</i> (1973)
Apple	Scab (<i>Venturia inaequalis</i>)	Rousselle <i>et al.</i> (1974)
Apple	Woolly aphid (<i>Eriosoma lanigerum</i>)	Knight <i>et al.</i> (1962)
Potatoes	Potato viruses X and Y	Howard (1970)
Potatoes	Wart disease (<i>Synchytrium endobioticum</i>)	Howard (1970)
Tobacco	Tobacco mosaic virus	Troutman and Fulton (1958)
Tobacco	Wildfire and angular leafspot (<i>Pseudomonas</i> spp)	see page 190
Wheat	Hessian fly (<i>Mayetiola destructor</i>)	Gallun <i>et al.</i> (1975)
Wheat	Cereal leaf beetle (<i>Oulema melanopus</i>)	Webster (1977)
Alfalfa	Spotted alfalfa aphid (<i>Therioaphis maculata</i>)	Nielson and Don (1974)
Oats	<i>Helminthosporium</i> spp.	Caldwell (1968)
Cabbage } Tomatoes } Peas }	<i>Fusarium oxysporum</i>	Walker (1966)

The use of some race-specific major resistance genes has created serious problems for plant breeders and farmers (Marshall, 1977). However, major genes can play an important part in the control of pests and diseases, and many condition resistance that has been durable (Table 13.2). Some workers, for example Person, Groth and Mylyk (1976), consider that polygenic control of disease resistance may be no more stable than monogenic resistance. However, although some polygenic resistance systems are known to be race-specific, the majority of such systems have been durable. Even those genes that control

transient resistance can be managed in a number of different ways so that they contribute significantly to pest or disease control. For example, they can be incorporated into a single variety in appropriate combinations which confer greater durability (Wolfe and Barrett, 1977); alternatively they can be used in multiline varieties or mixtures of varieties, and their use can be controlled by geographic localization or recycling, as suggested by Person *et al.* (1976), Frey, Browning and Simons (1977), Groth and Person (1977), and Parlevliet and Zadoks (1977).

The Effectiveness of Different Types of Resistance

It is very important for breeders to produce varieties with durable resistance, so that they do not have to find and use new resistance genes to overcome the effects of resistance-breaking variants. The need to introduce a succession of new genes into breeding material is often carried out at the expense of genetic improvements in other desirable attributes, for example yield and quality. If plant breeders were able to identify and employ those types of resistance which confer durability, greater improvements in characteristics other than pest or disease resistance could be achieved from the same input of time, labour and other resources.

Certain types of resistance have been more durable than others. Types which help host plants to avoid pest and disease attack seem to have given a particularly effective and durable control. The resistance of barleys with closed flowers to loose smut (caused by the fungus, *Ustilago nuda*) is an excellent example of disease avoidance: the pathogen is unable to infect the ovary of closed-flower barleys because spores are excluded by a physical barrier; the fungus cannot therefore complete its life cycle. It is difficult to envisage how variants of *U. nuda* could circumvent this kind of disease-avoidance mechanism, which is therefore truly non-race-specific. Some apple varieties avoid infestation by several insect pests because their buds do not generally break until the hatching or emergence period of these insects has finished (Briggs and Alston, 1967). This kind of multiple pest avoidance also seems to be non-race-specific and durable and can greatly reduce pest damage.

A tendency to escape infection plays an important part in resistance to many pests and diseases. For example, tall-strawed wheat varieties tend to be more resistant to *Septoria nodorum* than semi-dwarf types, because long internodes reduce splash dispersal of the spores from leaf to leaf (*see* page 55). Leaves from some wheat varieties collect fewer spores of *Puccinia striiformis* from a spore cloud than do those of other varieties, and are less affected by yellow rust as a result (*see* page 56). Aphid vectors of some viruses tend to walk off plants of some raspberry varieties and do not feed on them long enough to transmit viruses or to reproduce (*see* page 366). Such non-preference (or non-acceptance) resistance to insects is generally less race-specific than most other types of resistance, including antibiosis (Webster, 1977).

Resistance to the establishment of a pathogen, which is often manifested as hypersensitivity in the case of fungi, bacteria and viruses, can give a spectacular disease control and is usually easy to manage in breeding programmes; for these reasons this type of resistance has been widely used in many crop

species. Although hypersensitivity has often proved to be highly race-specific, other forms of resistance to pathogen establishment, such as extreme resistance to potato virus Y (see page 235) are (by definition) non-race-specific.

Antibiosis, which causes a reduction in the growth or reproduction of a pest or pathogen, can involve several distinct mechanisms, some of which are highly race-specific. Antibiosis can be based, for example, on the production by the host plant of a toxic compound, which may be either pre-existing or produced in response to parasite attack, or it may be based on the absence or low availability of some compound which is essential for the parasite concerned.

Tolerance has been an important characteristic of many resistant varieties, particularly with viruses and insect pests. Although there are several theoretical disadvantages of using some types of virus tolerance (see page 225), tolerance has reduced damage by many pests and diseases in a wide range of crop plants. Tolerance to insect pests does not select its own biotypes and is, therefore, apparently non-race-specific (Gallun, 1977); resistance-breaking problems have not generally been encountered with tolerance to virus diseases.

Different types of resistance therefore confer different levels of control and of durability. Complex types of resistance, and combinations of different types of resistance, can be expected to give a more complete and durable control of parasites than one simple type. For example, a resistant variety that expressed disease-escape, hypersensitivity, antibiosis and tolerance to a pathogen would probably give an excellent and durable disease control. On the other hand, such a combination might be very difficult to select for, and manage, in a breeding programme.

Some General Inferences

Resistant crop plant varieties have been developed against all the major groups of the parasites that attack them, including fungi, bacteria, mycoplasmas, viruses, invertebrate and vertebrate animal pests and parasitic weeds. Success has been achieved with both obligate and facultative parasites and with specialized and unspecialized parasites. Plants of some crop species are known to be resistant even to locusts and to some bird pests, which will feed on many different kinds of plant material.

Varieties with multiple resistance to several pests and diseases have been produced in many crops. For example, a new Japanese soya-bean variety, Okushirome, is resistant to at least two fungal pathogens (*Spaceloma glycines* and *Cercosporina kikuchii*), to two viruses (soya-bean mosaic and stunt viruses) and to a nematode, *Heterodera glycines* (Matsumoto *et al.*, 1974). The strawberry Cardinal shows good resistance to several fungal diseases, including *Mycosphaerella fragariae*, *Diplocarpon earliana*, and *Sphaerotheca meularis*, and tolerance to the two-spotted mite, *Tetranychus urticae* (Moore, Bowden and Sistrunk, 1975). High-yielding rice varieties with resistance to five insect pests, including *Nephotettix virescens*, *N. nigropictus* and *Niliparvata lugens*, and to bacterial blight and tungro virus, have been developed (Prakasa Rao and Sastry, 1975).

Host-plant resistance is not confined to the growing crop, and varietal differences in susceptibility to pests of stored crops have been reported in several crop species. For example, Chahal and Singh (1974) found that the grain of

some wheat varieties is much more susceptible to the rice weevil, *Sitophilus oryzae* and the lesser grain borer, *Rhizopertha dominica*, than are other varieties. Varietal differences in resistance to *S. oryzae* have also been observed in maize (Van Schoonhoven *et al.*, 1975). These results show that there is great potential for the breeding of crop varieties that would be resistant to post-harvest pests and diseases, which cause enormous losses of yield throughout the world. Success in breeding varieties resistant to non-parasitic diseases, such as manganese toxicity in soya-bean (Carter, Rose and Reading, 1975), emphasizes the enormous scope of breeding for resistance in raising the level of crop production.

Resistance to most pests and diseases has been found without great difficulty whenever it has been sought with determination. The resistance has sometimes been expressed as near-immunity, but more often has given only a partial control, so that resistant varieties can be badly damaged in severe epidemics. For this reason, resistant varieties have usually been used in conjunction with other control methods as part of an integrated control programme.

Most sources of resistance to pests and diseases have been found in existing cultivated varieties, from which it has been fairly easy to transfer the resistance to new varieties. In other cases resistance has been discovered, either in uncultivated plants or land races of the same species, or in related species or genera. As Harlan (1977) pointed out, resistance to disease is the normal condition and good sources of resistance are often 'near at hand'.

The first step in breeding for resistance has generally been to screen locally adapted varieties, using empirical techniques, in glasshouse or field tests. Where no resistant plants could be recognized in these local varieties, exotic varieties and related wild species were screened. The usual practice has then been to cross the resistant plants with plants of advanced breeding material, the progenies of succeeding generations being tested and selected for resistance and other desirable traits. These general procedures have been employed in breeding for resistance to all types of pests and diseases, although the detailed methods used have differed according to the type of parasite and the breeding system of the host plant concerned.

Successful selection for resistance to most pests and diseases has been carried out both in the field and in the glasshouse, using either artificial or natural infection. The majority of resistant varieties have been developed without any clear understanding of the genetics or mechanisms of resistance that are involved. Where these are understood, the knowledge has usually been gained retrospectively, that is, after the resistant varieties have been bred. This information has, therefore, helped us to understand what has happened during the process of breeding for resistance, rather than to provide direct guidance to the breeder in developing resistant varieties. Although a detailed understanding of the genetics of resistance and of the resistance mechanisms would undoubtedly help plant breeders in the future, the breeder has had in the past to rely mainly on empirical methods in breeding for resistance. These empirical selection and breeding techniques have been enormously successful and have been used very effectively to develop many varieties of all the major crop species with resistance to a wide range of pests and diseases.

Plant breeders have often been criticized because the resistance of many of their varieties has been overcome by new variants of parasites. The problems of race specificity have been greatly exaggerated, however, and there have been

very few catastrophic pest or disease attacks as a result of a 'breakdown' of resistance. Several forms of resistance that are known to be highly race-specific have given a worth-while protection in the field for many years. There are many examples of durable resistance involving race-specific major genes (Table 13.2), some of which have been described in earlier sections of this book. For example, combinations of race-specific major genes for resistance to *Puccinia recondita* in wheat are still effective in controlling leaf rust in many parts of the world, in spite of the presence of corresponding virulence genes in pathogen populations (see page 101). Race-specific resistance has also given a good control of *Fusarium* wilt in tomato for many years (see page 136).

Resistance-breaking variants have been a serious problem with some parasites, particularly certain fungal pathogens. Although examples of race specificity, where there is a gene-for-gene interaction between host and pathogen, are known in each of the major groups of pests and pathogens, resistance breaking has generally been less important with bacteria, viruses and animal pests than with fungal pathogens. However, variation in very many parasites has greatly complicated breeding programmes and has been responsible for the withdrawal or unpopularity of many high-yielding varieties. Genetic variation of parasites has, therefore, often been less important to farmers than to plant breeders, who have had to spend valuable time, effort and resources on trying to overcome race specificity, rather than concentrating on improving yield and quality.

Race specificity is associated with certain types of resistance, particularly with hypersensitivity, and with simply inherited corresponding genetic systems in host and parasite. Resistance controlled by single genes has often been associated with problems of race specificity. Marshall (1977) has stated that one of the major factors leading to calamitous losses from disease in the past has been the widespread use of single genes for resistance over vast areas. However, he also points out that alternatives to the use of single resistance genes are relatively laborious and demanding of the plant breeder's time and resources. There are, therefore, good reasons why major resistance genes should continue to be used in future breeding programmes. A resistance gene *Pa*₇, in combination with certain minor genes, conditions resistance to leaf rust in barley that has been, and still is, effective everywhere in the world (Parlevliet and Kuiper (1977). Rice varieties carrying the major genes *Xa*₁ or *Xa*₄ for resistance to *Xanthomonas oryzae* have expressed a high level of resistance to bacterial blight for many years in most areas where they have been grown (Khush, 1977).

The presence of major resistance genes can mask the presence of minor genes that might help to increase the durability of resistance (e.g. Parlevliet and Kuiper, 1977). When such masking occurs, selection for durable resistance is impeded and there is a tendency for the minor genes to be lost without positive selection. There are many examples in the literature of such losses of 'field resistance'. For example, many normally resistant rice varieties have been even more badly damaged by blast disease than susceptible varieties, when their major-gene resistance has been overcome by new races of *Piricularia oryzae*; Kiyosawa (1977) has called this condition a 'super-breakdown'. This corresponds with the 'Vertifolia effect' in potatoes which has been described by Van Der Plank (1968). Robinson (1977a, b) considers that it is impossible to measure horizontal resistance, or to accumulate genes which control horizontal resistance, if vertical resistance genes are present and are operating. He

therefore suggests that vertical resistance (or its effects) must be eliminated before plants can be effectively selected for horizontal resistance. This strategy has been adopted by Bingham (1975) in selecting wheat for resistance to yellow rust (*Puccinia striiformis*); resistant seedlings in segregating populations are discarded because they apparently express major-gene race-specific resistance and the remaining plants (or their progeny) are then re-tested for resistance to yellow rust as adult plants. Plants which are resistant as adults are then selected in the hope that they express durable resistance. However, adult-plant resistance can be highly race-specific (Johnson and Bowyer, 1974), and the discarding of resistant seedlings from a population does not therefore guarantee that the resistance of surviving plants will be durable.

Minor resistance genes are not always lost in the presence of major genes, even when there is no deliberate attempt to retain them. For example, a few barley varieties express durable, partial resistance to powdery mildew (*Erysiphe graminis*) when their major-gene resistance has been overcome by resistance-breaking races. In the UK, Vada and Deba Abed have maintained a reasonable degree of field resistance to powdery mildew for more than 10 years although virulent races of *E. graminis* have been widespread (Moore, 1977).

Many crop varieties have continued to express a similar high level of resistance to a parasite, even when they have been exposed to that parasite for many years and over a large area. For example, adult plants of the wheats Little Joss and Atle are as resistant to *Puccinia striiformis* today as they were when they were first released many decades ago (Lupton and Johnson, 1970). However, the fact that races of *P. striiformis* that can attack these varieties have not been found, does not necessarily mean that such races will never occur. Some varieties with proved durable resistance have eventually succumbed to some variants of a parasite. For example, the apple Winter Majestic (= Northern Spy), which has been very resistant to the woolly aphis (*Eriosoma lanigerum*) since its introduction in 1831 (Pathak, 1970), has recently been attacked by a new biotype of *E. lanigerum* in Australia (Sen Gupta and Miles, 1975).

Some Benefits and Drawbacks of Breeding for Resistance

Many examples of successful programmes of breeding for resistance to a wide range of parasites, in most of the major crop species, have been given in this book. These programmes have culminated in the production of a large number of resistant varieties, many of which have been extensively grown and have contributed significantly to the control of pests and diseases. It is difficult to quantify the financial benefits which have resulted from the use of resistant varieties, but some potentially important pests and diseases have been rendered economically unimportant solely because resistant varieties have been widely grown. For example, maize rust is no longer a damaging disease in the USA, mainly because this disease is controlled by resistant varieties that express a durable resistance to *Puccinia sorghi*, conditioned by polygenes (Hooker, 1973). Growing Hessian fly-resistant wheat varieties has caused the virtual disappearance of this pest from large areas of the USA (see page 325). The wheat stem sawfly was a very serious pest in parts of North America until solid-stemmed resistant varieties were grown on a large scale; the resistant wheat Rescue is estimated to have saved farmers in Montana more than \$40 million over a period of 10 years (see page 328). In these and several other examples, resistant varieties

have given an almost complete control of the parasites concerned. However, resistant varieties usually give only a partial control, but even partially resistant varieties can make all the difference between profit and loss or, in less developed countries, the difference between plenty and starvation. Partially resistant varieties can be severely damaged in serious epidemics but other control measures are usually much more effective on partially resistant varieties than on susceptible varieties.

The economic benefits of resistant varieties are therefore very great but there are also other important advantages in breeding for resistance. As Pathak (1970) has pointed out, resistant varieties reduce pest damage at all levels of infestation, do not cost more to grow in terms of time and labour than susceptible varieties, are generally non-toxic to man, livestock and wildlife, and do not pollute the environment.

Plant breeding for pest and disease resistance is a slow and expensive process and it has often taken 10–15 years to develop agronomically acceptable resistant varieties, even when sources of resistance were readily available. New techniques, including the artificial shortening of the breeding cycle of crop plants, have greatly reduced the number of years which are necessary to produce a new variety. The development, manufacture and distribution of new pesticides can take even longer, and cost much more, than the breeding of a new variety. Resistant varieties can generally be maintained at very small extra cost, whereas new supplies of pesticides have to be manufactured each year.

The need for more and better resistant varieties will increase as pesticides become more and more expensive to develop and distribute. It costs many millions of US dollars to develop a new pesticide and every new compound has to undergo stringent tests for toxicity to organisms other than the target organism. In view of the very considerable expense involved, there will be an increasing tendency for agrochemical manufacturers to develop pesticides with a very broad spectrum of biological activity, because it will be uneconomic to produce new compounds which are effective against only one target parasite. Such broad-spectrum pesticides often affect many non-target organisms and can upset the ecological balance in treated environments. Chemical control methods can impede other control measures by killing beneficial organisms, for example predators or hyperparasites of pests or pathogens of crop plants. On the other hand, host-plant resistance is usually effective only against the target organism. In many situations, however, resistant varieties will not by themselves give an adequate level of pest and disease control, and it will then be necessary to apply broad-spectrum pesticides in an integrated control programme, as suggested by many workers, including Way (1974) and Haskell (1977).

References

The references cited in this chapter, together with those for Chapter 14, are listed in References – Part VI, pages 441–444.

FUTURE PROSPECTS

Breeding Objectives and Priorities

Breeding for resistance has been very successful in reducing damage caused by many pests and pathogens ranging in complexity from viruses (the simplest pathogens) to higher plant and vertebrate parasites. There is no reason to suppose that breeding for resistance to other parasites, including bird and mammal pests and insects with wide host ranges, would be any less successful. A search for sources of resistance to all pathogens and pests of major crop species is therefore probably worth while, even if these sources are not exploited immediately by plant breeders; they would then be available for use at short notice when necessary.

Although the introduction of resistance to several different pests and diseases into a single variety can be difficult and time-consuming, the difficulties are not insuperable. This book contains many examples of varieties with 'multiple' pest and disease resistance (*see* page 429). The production of such varieties is an important breeding objective because there is little advantage to the farmer in growing a variety that is resistant to one insect pest if insecticides have to be applied in any case as a routine to control other insects. Sources of multiple resistance are not uncommon. For example, Harlan (1977) reported that a wheat (Accession No. PI 178383), which was collected from eastern Turkey in 1948, expresses high levels of resistance to yellow rust (*Puccinia striiformis*), bunt (*Tilletia* spp.), flag smut (*Urocystis* spp.) and snow mould (*Fusarium* spp. and *Typhula* spp.). Such accessions are particularly valuable because it is then not necessary for the plant breeder to introduce resistance to each parasite separately. Duvick (1977) has emphasized the urgent need for developing, maintaining and using more germplasm collections on a world-wide scale. Such collections would provide important sources of multiple pest and disease resistance.

It has been the general experience that the growing of near-immune varieties of crop plants imposes a very great selection pressure in favour of resistance-breaking variants of the parasites concerned. It may therefore be more sensible to concentrate on the development of partially resistant varieties, which reduce the effects of parasitism to an acceptable level, rather than to attempt to breed

near-immune varieties. It is particularly important that plant breeders do not produce varieties of any crop that are very susceptible to any important, or potentially important, pest or disease. Although this approach involves the testing of breeding material against a wide range of parasites so that very susceptible plants can be discarded, it would do much to avoid calamitous pest and disease outbreaks in the future.

The problems of race specificity and transience of resistance have been stressed many times in this book (see page 92 for example), although their seriousness may have been exaggerated in the past. Where resistance-breaking variants of a parasite become a severe problem, attempts should be made to identify more durable types of resistance. However, it is not easy to distinguish between durable and transient types of resistance before the resistance has been exposed for many years to the parasite concerned. Without detailed information concerning the nature of resistance, the plant breeder cannot predict whether a particular type of resistance will be durable, although the chances of durability will be increased if only those parents with proved durable resistance are used in a breeding programme. Until there is a better understanding of the underlying mechanisms which contribute to durability, breeders must rely on the empirical incorporation of different types of resistance or different resistance genes, in the hope that this will provide greater durability.

An alternative approach would be to manage forms of transient resistance so that the effects of resistance-breaking are minimized, for example by using multiline varieties or varietal mixtures (e.g. Frey *et al.*, 1977; Groth and Person, 1977; Wolfe and Barrett, 1977). It often seems, however, that more effort has been expended in trying to find ways of living with the problems of race specificity than in trying to avoid them by breeding for durability. A high priority should be given to more work on horizontal or durable resistance.

In breeding for resistance, plant breeders must allocate priorities to particular pests and diseases, because it is not possible to select simultaneously for resistance to all the pests and diseases that can attack their particular crop. It is often difficult to decide whether priority should be given to those parasites which (1) are potentially the most damaging, but which can usually be adequately controlled by other measures, (2) are less damaging but are difficult or expensive to control, or (3) occur only sporadically or locally, so that other control methods are not usually applied. The plant breeder concerned, with the help of his specialist advisers, will usually be in the best position to decide these priorities, but there are many complicating factors.

In countries with a well-developed agriculture, most pests and diseases are generally kept in check to some extent by a series of integrated control procedures, including the widespread and liberal use of expensive pesticides. In such situations, pest and disease damage will not usually be of overriding importance, and breeding for resistance must not be at the expense of increasing yield and quality through improved varieties; resistance will then be only one of several desirable attributes. In developing countries, however, the potential yield of a new variety is often irrelevant because of local financial and other constraints, the most important of which are attacks by pests and diseases which can reduce potential crop yields by more than one-half. In such circumstances, resistance becomes the most important breeding objective, particularly when effective pesticides are too costly for general use and resistant varieties may be the only practical control measure.

The Dangers of Genetic Uniformity

The potential dangers of too much genetic uniformity or homogeneity in crop plants, in connection with susceptibility to pests and diseases, have been recognized for many years (Committee of National Research Council, 1972). The dangers were first highlighted by a severe epidemic of Victoria blight disease (*Helminthosporium victoriae*) on oats in the USA during the 1940s. Closely related hybrid varieties accounted for more than 80 per cent of the oat acreage in the USA in 1945 and these were all severely attacked by *H. victoriae* in 1946 and 1947. The lack of genetic diversity in the form of varieties of different origin meant that almost the entire oat crop was at risk from the disease, and very great losses of yield occurred. Fortunately, it was possible to replace these varieties in subsequent years with cultivated varieties that were based on a resistant variety, Bond (Browning, 1972). A similar situation arose in the maize crop in the USA in the early 1970s. More than 80 per cent of the maize crop in 1970 was planted with varieties with the same type of cytoplasm, which carried extra-chromosomal genes conferring male sterility. These varieties were badly damaged in 1970 and 1971 by an epidemic of southern corn leaf blight, caused by race T of the causal fungal pathogen *Cochliobolus heterostrophus* (*Helminthosporium maydis*). Although a recurrence of this epidemic was prevented by growing other maize varieties, these catastrophic outbreaks of southern corn leaf blight over such a large area would not have happened if several genetically and cytoplasmically distinct maize varieties had been grown.

There are dangers of similar epidemics of pests and diseases in many crops where there is a lack of genetic diversity (Marshall, 1977). Most of the major crops in the USA comprise very few distinct varieties (Macer, 1972) and there is a trend towards fewer and more uniform varieties. For example, about 80 per cent of the US cotton crop in 1976 was planted with only six varietal types, all of which are susceptible to many potentially damaging diseases and insect pests (Duvick, 1977). Four varieties dominated the hard red winter wheat acreage in the USA in 1976. There is a similar genetic uniformity in barleys that are resistant to barley yellow dwarf virus (*see* page 264), because all sources of resistance except one have descended from a narrow range of Ethiopian barleys (Qualset, Prato and Vogt, 1977; Schaller, 1977). In sugar beet, the older, heterogeneous varieties have been almost completely replaced throughout the world by mono-germ varieties, which not only share a similar genetic base but also have a common cytoplasm carrying male-sterility factors. Frankel (1977) points out that this situation in sugar beet closely parallels that which occurred in maize in the USA in the early 1970s. Most of the modern semi-dwarf wheats that are so extensively grown throughout the world, have been partly derived from a Japanese variety, Norin 10. Is there not, therefore, a danger of disastrous pest and disease outbreaks in cotton, sugar beet and wheat because of this genetic uniformity?

These dangers of genetic uniformity can largely be avoided if plant breeders use different sources of genes and cytoplasm in their breeding material. It will be very important to achieve sufficient genetic diversity in all crops so that epidemics, such as those of Victoria blight in oats and southern corn leaf blight in maize, do not occur in any crop. It would certainly be dangerous to rely too much on any one individual source of resistance to a disease or pest in developing future varieties.

Conservation of Germplasm

The 'gene pool' of genetic diversity of crop species consists of primitive varieties (land races), recently developed varieties and wild relatives (Frankel, 1977). Land races have been particularly valuable sources of resistance in most of the major crop species. For example, many thousands of local rice varieties have been collected from all the main rice-growing areas, and many valuable sources of resistance to pests and diseases have been found amongst them (Ou, 1977; Pathak, 1977). Varieties from one region have often been useful to plant breeders in others. For instance, an Indian variety, TKM6, has been used as a source of resistance to the brown planthopper (*Nilaparvata lugens*) in the Philippines (see page 347); *Oryza nivara*, also from India, has been one of the main sources of resistance to grassy stunt disease (see page 206). Tatep from Vietnam has been used in many countries as a source of resistance to *Piricularia oryzae*, the blast disease fungus (see page 111).

An extensive collection comprising more than 20000 cultivated and wild barleys has been assembled and maintained by the United States Department of Agriculture (USDA) at Beltsville, Maryland, and many important sources of disease resistance have been found in it (Schaller, 1977). The USDA Plant Introduction Service has been responsible for the world-wide collection and preservation of varieties and wild relatives of many other crops, including alfalfa (Stanford, 1977). More than 2200 accessions of wild wheats (*Triticum* spp.) and their near relatives, including *Aegilops* spp., which were collected from the Middle East and Asia Minor, are kept at the University of California at Riverside (Johnson and Waines, 1977); resistance to the 'take-all' fungus, *Gaeumannomyces (Ophiobolus) graminis*, to which no good sources of resistance are known, and to several nematode and insect pests, is being sought in this material.

These artificial gene pools, which can be preserved in collections as seeds, vegetative parts or tissue cultures, will provide most of the essential ingredients for pest- and disease-resistance breeding in the near future. However, natural gene pools are being depleted at an alarming rate by modern agricultural methods, including the widespread use of modern varieties and herbicides; many wild species and land races have already been lost. New and more extensive collections of germplasm of all crop species are urgently needed so that genetic diversity can be artificially maintained. The natural gene pools are the result of evolution over thousands of years involving spontaneous mutations, genetic recombination and natural selection for resistance to pests and diseases; they will be impossible to replace.

New Genetic and Breeding Techniques

There is an abundance of genetic variability in existing land races, varieties and wild species for present breeding purposes, although it is obviously not possible to test every genotype for the presence of potentially useful resistance genes. Induced mutations can supplement this diversity in such a way that the chance of finding rare genes or alleles is increased; they are likely to become an increasingly important source of genetic variability (Nilan, Kleinhofs and Konzak, 1977). Mutations can be induced either by physical means (X-rays, gamma rays or neutrons) or by chemical mutagens which include ethyl methanesulfonate

(EMS), various nitroso compounds and sodium azide. Physical methods generally cause a high frequency of chromosome breakage, which is associated with gross genetic aberrations. Chemical mutagens, particularly sodium azide, are more specific in their actions and cause a high proportion of mutations at individual gene loci (Nilan *et al.*, 1977).

Many induced mutants with improved disease resistance have been identified in a wide range of crops, including resistance to powdery mildew in barley, and to yellow, leaf and stem rusts in wheat (Sigurbjörnsson and Micke, 1974). Induced barley mutants with recessive alleles at the *ml-o* locus on chromosome 4, which conditions resistance to powdery mildew (*Erysiphe graminis*), were identified in the 1960s in Denmark (see page 104) and this resistance has been effective whenever it has been tested (Jørgensen, 1977). Although the *ml-o* gene has since been identified in natural barley populations, induced mutations have enabled this gene to be introduced into several modern barley varieties. This ability to induce mutations in existing varieties has great advantages over the introduction of resistance genes by conventional breeding techniques. The transfer of resistance genes from alien genotypes frequently results also in the transfer of undesirable genetic material.

Induced mutations increase the frequency of mutants which occur spontaneously, but they do not create completely new genetic variability. As more-refined techniques are developed, induced mutations will become increasingly important in breeding for resistance, as natural sources of genetic diversity become depleted. The possibilities of mutation breeding for pest and disease resistance have, so far, been explored on a very limited scale, and further work is clearly justified.

Several cytogenetic techniques can be employed to introduce resistance genes from one genotype into others. For example, chromosomes carrying resistance genes to wheat yellow rust (*Puccinia striiformis*) have been transferred to wheat from alien species, including *Aegilops comosa* (Riley, Chapman and Johnson, 1968). A single chromosome, designated 5BS-7BS, which carries genes that condition durable resistance to yellow rust, is being transferred from Hybride de Bersée to other wheats (Johnson and Law, 1975) using aneuploid stocks developed by methods described by Law and Worland (1973). Such techniques will enable breeders to introduce resistance genes into breeding material more quickly and surely than by most other techniques.

New methods of genetic engineering, many of which are now being developed with micro-organisms, could eventually be applied to crop plants. For example, genetic material could be transferred asexually between sexually incompatible species by means of organelle transfer, protoplast fusion or the transfer of foreign DNA (Polacco and Polacco, 1977). These techniques would allow alien resistance genes to be transferred to crop plants from plants which are outside the normal host range of a parasite, in a way that would be impossible by more conventional methods; such resistance would probably be truly non-race-specific.

The Future Role of Resistant Varieties

Resistant varieties will probably play an increasingly important part in the control of crop pests and diseases. The escalating cost of pesticides, the spread of pesticide-resistant variants, and the possibility of unacceptable chemical residues

in food plants, will make alternative control methods, such as host-plant resistance, even more attractive in the future. Chemical control measures can interfere with pest/host-plant ecosystems which, in turn, can cause problems with secondary pests (Haskell, 1977). Resistant varieties are usually free from such problems.

It would be very unwise to rely solely on any one measure for the control of any pest or disease, whether this is host-plant resistance or a chemical control method. It is now possible to use resistant varieties in integrated control programmes, which may also include the minimum use of pesticides, the improvement of plant sanitation and other methods of biological control (Browning, 1972). Haskell (1977) has stressed the dangers of a 'single component' system of pest control, such as the use of broad-spectrum pesticides for a long period: he cites two examples of disasters which followed such an approach; the devastation by pests of the cotton crop in Peru, and of the cocoa crop in Sabah. The situation began to improve only when spraying with pesticides was completely abandoned. Nevertheless, pesticides will continue to be a very important control method and will support other cultural and biological control measures. Resistant varieties have been so successful that they must be the cornerstone of any integrated control programme for most pests and diseases in all crops.

New varieties with an adequate level of resistance to almost any pest or disease can be developed in all the major agricultural and horticultural crops within a few years, provided that sufficient resources, in terms of skilled manpower, facilities and funds, are provided for this vital work. No investment will pay greater dividends in raising the standard of living and health throughout the world than the further development of resistant crop varieties.

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